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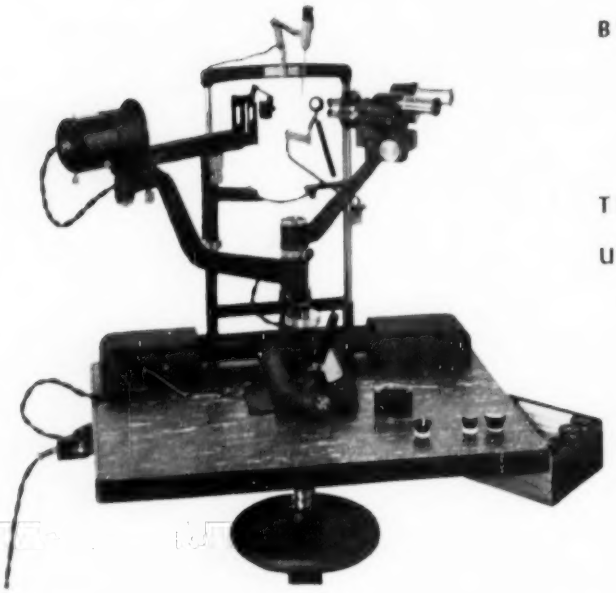

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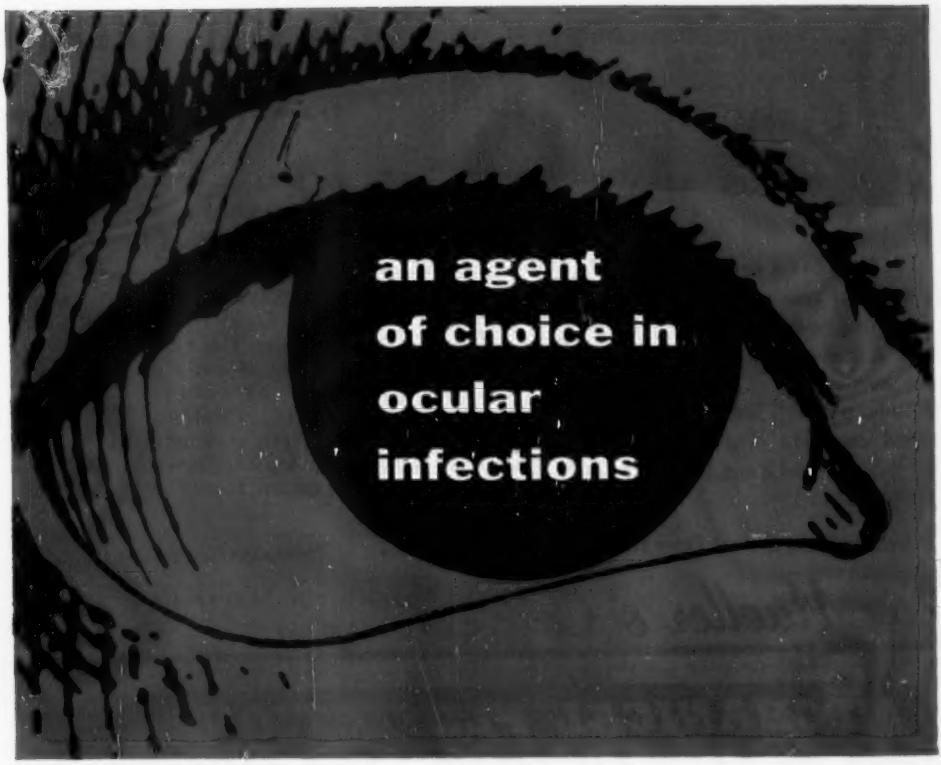

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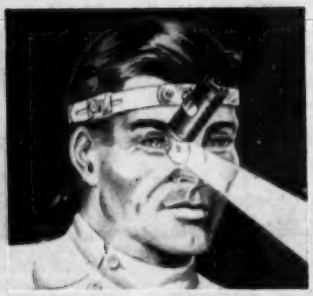

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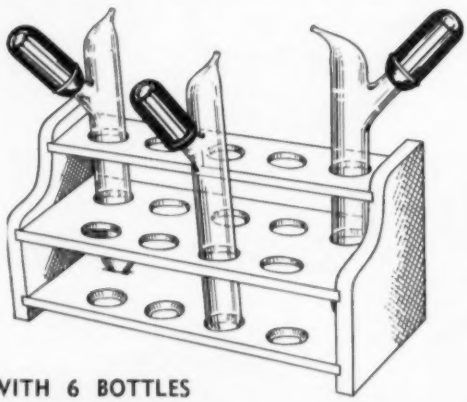

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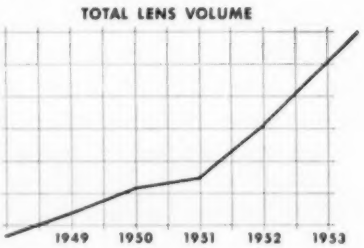

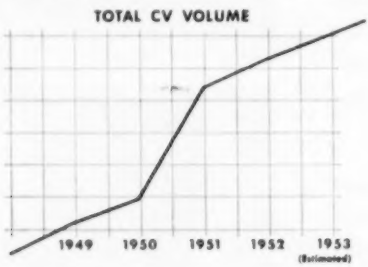

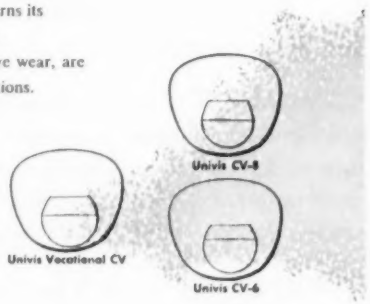

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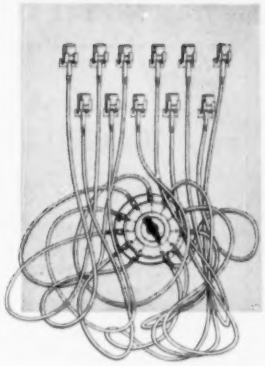

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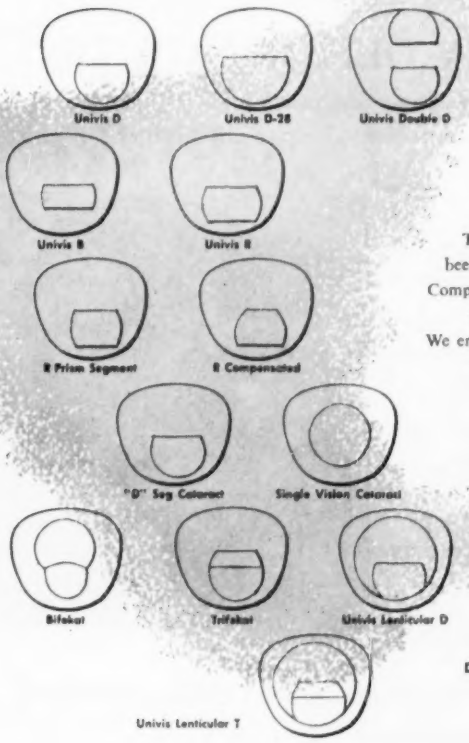

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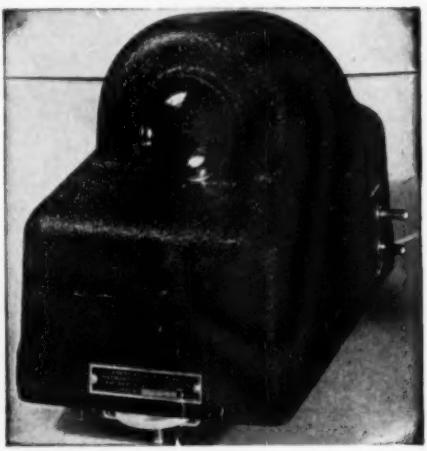

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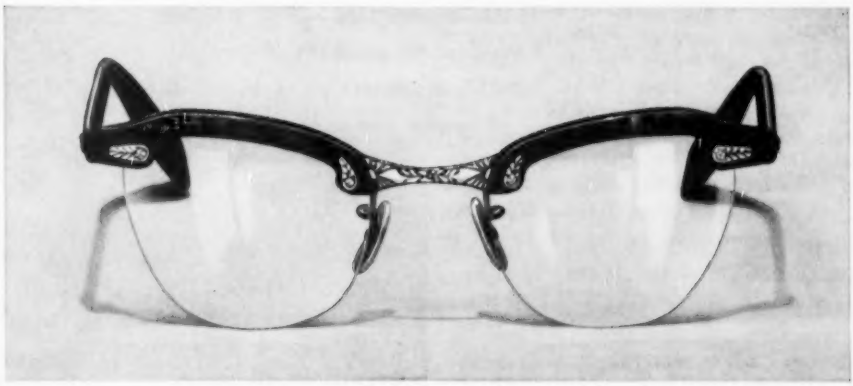

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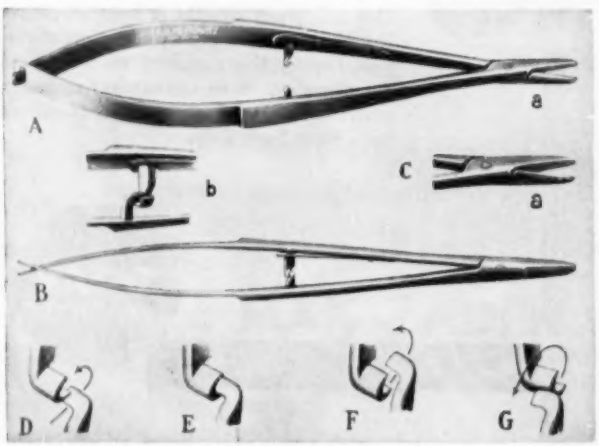

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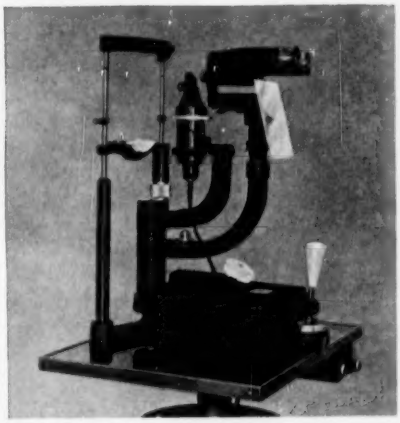

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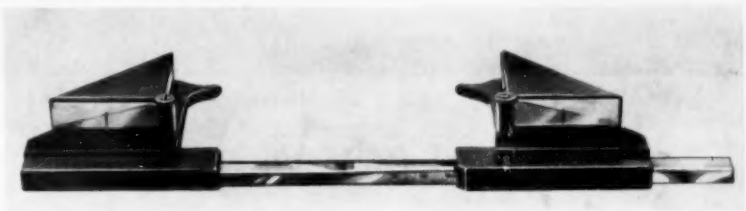

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ASCORBIC ACID IN AQUEOUS HUMOR AND SERUM OF PATIENTS WITH AND WITHOUT CATARACT

Physiologic Significance of Relative Concentrations

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DETROIT

MANY ATTEMPTS have been made to explain how the concentration of ascorbic acid in the aqueous humor is maintained in excess of that in the blood.[1] Three different mechanisms have been proposed: (1) Ascorbic acid is synthesized by the lens *; (2) ascorbic acid is transported across the ciliary epithelial border in the oxidized state (i. e., as dehydroascorbic acid), where it is reduced to ascorbic acid by the lens †; (3) ascorbic acid is selectively concentrated by the ciliary epithelium, whence it diffuses to the posterior chamber.[7]‡

The chief evidence in support of the theories that associate the lens with the maintenance of relatively high concentrations of ascorbic acid in the aqueous humor is the observation that the concentration of the acid is less than normal when the lens is cataractous or absent. For instance, reduced concentrations of ascorbic acid in the aqueous humor have been observed in patients with senile cataract, in eyes of animals with experimental cataract, and in eyes of rabbits from which the lenses had been removed.§

Other investigators found, however, that the concentration of ascorbic acid in the aqueous humor of eyes with cataractous lenses was less than normal only in about half of the patients examined,[11] and that normal concentrations were obtained in aqueous humor of young patients with aphakic eyes.[12] More recently, one of us (V. E. K.) [8] reported that as long as there were no signs of inflammation in aphakic eyes of rabbits the concentration of ascorbic acid in the aqueous humor was at least as great as that in the other eye of the same animal whose lens had not been removed. However, because of the technical difficulty of removing lenses from the eyes of rabbits it was possible that some lens tissue still remained in the "aphakic" eye.

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* Fischer,[2] Müller and Buschke.[3]

† Müller and Buschke.[3] Goldmann and Buschke, references 4, 5, and 6.

‡ Evidence in favor of the third hypothesis is only indirect; for example, Kinsey [8] has shown that the concentration of ascorbic acid in the aqueous humor of the posterior chamber of rabbits is about one and a third times that in the anterior chamber. This observation is contrary to expectation were the acid to enter the posterior chamber as dehydroascorbic acid to be reduced by the lens.

§ References 3, 9, and 10.

Kronfeld [13] also concluded that the aqueous humor of aphakic eyes contains normal concentrations of ascorbic acid. He inferred the concentration of ascorbic acid from analysis of fluid from filtering blebs of patients having complete surgical aphakia. His results, too, are subject to criticism from a technical standpoint, since it is possible that for one reason or another the concentration of ascorbic acid which he found in the bleb fluid may not be the same as that in the aqueous humor. Thus, it can still be maintained that a normal concentration of ascorbic acid in aqueous humor is dependent upon the presence of normally viable lens tissue.

In view of the possibly inconclusive nature of the experiments involving aphakic eyes, we believed it worth while to reinvestigate the question of the comparative concentrations of ascorbic acid in the aqueous humor of patients with and without cataracts. Previous investigators who determined the concentration of ascorbic acid in the aqueous humor of patients with and without cataractous lenses did not make simultaneous determinations of the concentration of ascorbic acid in the blood. Since it is possible that patients with cataractous lenses may have been on dietary regimens containing more or less ascorbic acid than patients selected for controls, the validity of the conclusions with regard to the question of whether the lens was responsible for the excess ascorbic acid in the aqueous humor remains in doubt. The information which is required to resolve the issue is a knowledge of the ratios of the concentration of ascorbic acid in the aqueous humor to that in the serum. The present paper is concerned with presenting the results of an investigation which provides such information.

METHODS

Patients were prepared in the usual manner for intraocular surgery. The eyes of the first two patients (B. B. and F. H.) were anesthetized by retrobulbar injection of 4% procaine hydrochloride with epinephrine and hyaluronidase. A No. 27-gauge needle attached to a calibrated 250 μl pipette was introduced into the anterior chamber at the limbus; aqueous humor was transferred into the pipette by intraocular pressure. When most of the aqueous humor from the eye had entered the pipette, the needle was withdrawn and the eye was permitted to rest for approximately 20 minutes to allow re-formation of the anterior chamber prior to extraction of the cataract. Since the eyes were undesirably soft for operation at this time, a different procedure was used for the remainder of the patients.

Aqueous humor was withdrawn from the eyes of other patients under topical anesthesia and aseptic conditions on the evening before operation. The eyes of these patients had normal tension at the time of operation, the following morning.

After withdrawal, the aqueous humor was transferred to a small beaker, and aliquots of 50 μl were added to an equal volume of 8% metaphosphoric acid solution in a small test tube. Calibrated constriction pipettes were employed for delivery of samples. The solutions were mixed and placed in stoppered tubes in a refrigerator at 5 C.

In the first two patients blood was obtained by puncture of an antecubital vein eight hours after withdrawal of aqueous humor; in subsequent patients blood and aqueous humor samples were obtained concurrently. The blood was centrifuged, and 2.0 ml. of serum was mixed with 4.0 ml. of 4% metaphosphoric acid. The mixture was centrifuged, and measured quantities of the supernatant were titrated.

There were no borderline instances of cataract in the series of cases studied; the lenses in the cataractous series of patients were all sufficiently opaque to warrant extraction. In the control series of patients whose eyes were to be operated on for other pathologic conditions, the lenses were clear as viewed with a biomicroscope.

ANALYSES

Analyses of ascorbic acid were performed by titration in the usual manner with 2:6 dichlorophenolindophenol. The amount of dye required by blanks to produce an amount of color equivalent to the end-point selected was subtracted from all values. The dye was made up at a concentration of 80 mg. per 100 cc., filtered before use, and standardized with known concentrations of ascorbic acid made up in 4% metaphosphoric acid.

A Rehberg burette was used to deliver the dye for titration of aqueous humor, and a 1 ml. pipette graduated in hundredths of a milliliter was used for the serum

Concentrations of Ascorbic Acid in the Aqueous Humor and Serum of Patients with Cataracts, and Their Ratios

Patient	Age	Ascorbic Acid, Mg./100 Gm.		Ratio (Aq. H./ Serum)
		Aqueous Humor	Serum	
Patients with Cataractous Lenses				
B. B.	54	14.5	0.72	20.1
F. H.	78	4.9	0.24	20.5
C. Z.	56	18.8	1.75	10.7
M. F.	71	7.0	0.65	10.8
D. W.	48	13.3	0.58	22.5
K. Z.	70	19.5	0.98	19.9
J. B.	75	6.6	0.22	30.0
Average		12.1	0.74	19.2
Patients with Normal Lenses				
L. R.	56	12.6	0.98	12.9
P. H.	57	21.4	1.01	21.2
B. D.	39	20.0	0.70	28.5
Average		18.0	0.90	21.2

analyses. Samples of aqueous humor were analyzed in duplicate, and those of serum were analyzed in triplicate.

Preliminary tests showed that ascorbic acid is stable in 4% metaphosphoric acid at 5 C. for at least 36 hours; thus, no error was introduced in the two instances in which the titration procedure of aqueous humor was delayed for several hours. All of the blood specimens were prepared for titration within 30 minutes after withdrawal and were titrated within an hour.

Serum and aqueous humor were believed to be at a steady state with regard to ascorbic acid since none of the patients had any food containing appreciable quantities of ascorbic acid within six hours prior to withdrawal of aqueous humor and blood.

RESULTS

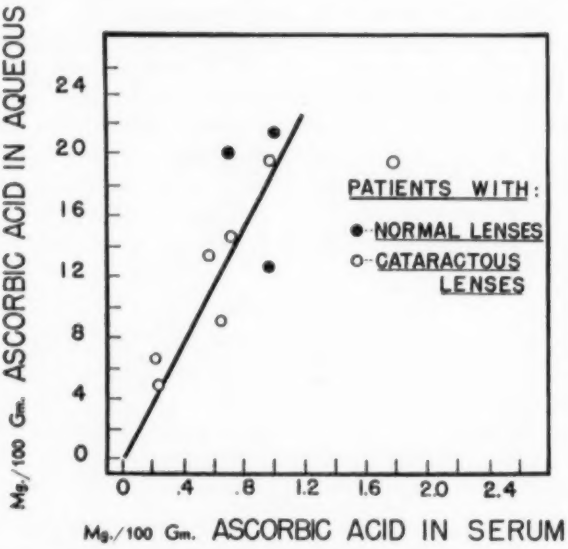
The data presented in the Table show that there is a wide variation in the concentration of ascorbic acid in aqueous humor and in serum among the various subjects. The average concentration of ascorbic acid in aqueous humor of the patients with cataract is less than that in the patients with normal lenses. However, because the concentration of the acid in the serum is also less in the group with cataracts the ratio of concentration in the aqueous humor to that in the serum is essentially the same for the two groups.

The Chart shows the relationship between the concentration of ascorbic acid in the aqueous humor and that in the serum in the patients with and without cataract. It appears that there is no difference and that the concentration of ascorbic acid in the aqueous humor increases with that in the serum.

COMMENT

The number of samples of aqueous humor of eyes of persons with normal lenses was small; however, the values obtained for the concentration of ascorbic acid both in the aqueous humor and in the serum fall within the range of values reported by others for adults with normal lenses.

If, as has been hypothesized previously, the lens is concerned with maintaining the concentration of ascorbic acid in the aqueous humor at approximately 20 times



Concentration of ascorbic acid in the aqueous humor and serum of patients with normal and cataractous lenses.

that of the blood, the present results suggest that the cataractous lens is as effective in doing so as the normal lens. In the introduction to this paper it will be recalled that the mechanisms, as previously proposed, by which the lens could maintain the excess ascorbic acid in the aqueous humor were (1) synthesis of ascorbic acid and (2) reduction of dehydroascorbic acid, which was assumed to be transported across the ciliary epithelial barrier into the posterior chamber.

Because it is well known that cataractous lenses have little or no metabolic activity and that the concentration of ascorbic acid, as well as other reducing substances, within the cataractous lens is negligible, it seems most improbable that a cataractous lens could synthesize ascorbic acid, or that it could reduce dehydroascorbic acid. Since, as was shown in the present study, there was little, if any, difference in the ratios of concentration in the aqueous humor to that in the serum

between the patients with normal lenses and those with cataractous lenses, it follows that the normal lens does not synthesize, or otherwise supply, ascorbic acid to the aqueous humor in any appreciable quantity.

The observation that the concentration of ascorbic acid in the aqueous humor varies with that in the serum is compatible with the belief that it is ascorbic acid in the reduced form that is secreted by the ciliary body, and, as in rabbits,[14] the rate of secretion is dependent, not on the presence of viable lens tissue, but on the concentration of ascorbic acid in the serum, at least up to a certain concentration in the serum.

It is of incidental interest that the ratio of the concentration of ascorbic acid in the aqueous humor to that in serum of human beings, i. e., approximately 20 to 1, is approximately that for rabbits.

The inference has sometimes been made that diminished concentration of ascorbic acid in the aqueous humor may cause cataract formation.[1] In view of the present observation that the aqueous humor of eyes with cataractous lenses contains a normal concentration of ascorbic acid, the therapeutic administration of vitamin C to patients with cataracts would appear to be irrational.

SUMMARY AND CONCLUSIONS

The concentration of ascorbic acid in the aqueous humor was found to vary approximately as the concentration in the serum, regardless of whether or not the lens was cataractous.

The results reported here, along with those presented in a previous paper,[8] make it seem highly improbable that the lens synthesizes appreciable quantities of ascorbic acid or that the lens reduces any dehydroascorbic acid assumed to enter from the ciliary body. They are in agreement, instead, with the idea that the major part, if not all, of the ascorbic acid present in the aqueous humor enters the posterior chamber from the ciliary body in the reduced form, and not as dehydroascorbic acid.

NOTE:—Since submitting this paper for publication, we had the opportunity of determining the concentration of ascorbic acid in the aqueous humor and serum of two patients with aphakic eyes. Four months previously, each patient had a lens removed by an intracapsular operation. The eyes of these patients showed no signs of inflammation or of lens remnants.

In the first patient the concentration of ascorbic acid in the plasma was 0.30 mg. per 100 gm. of water, and that in the aqueous humor of the aphakic eye was 6.8 mg. per 100 gm. of water. In order to assure a normal plasma concentration of ascorbic acid in the second patient, 400 mg. of ascorbic acid was given by mouth four hours prior to obtaining the samples of fluid. In this patient the concentration of ascorbic acid in the plasma was 1.05 mg. per 100 gm. of water, and in the aqueous humor, 14.8 mg. per 100 gm. of water.

The high ratio of the concentration of ascorbic acid in the aqueous humor to that in the plasma in eyes with cataractous lenses and aphakic eyes shows clearly that the mechanism involved in the production and maintenance of ascorbic acid in the aqueous humor is not dependent upon either the condition or even the presence of a lens.

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OCULAR ANTIBIOTIC-PRODUCING BACTERIA IN NORMAL EYES AND IN CONJUNCTIVITIS

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ANTIBIOTIC-producing strains of bacteria of the normal flora of various tissues of humans have been found to be very common in recent years. The intestinal tract,* eye,[4] nasopharynx,† skin and oral cavity ‡ have proved rich in such microorganisms. The antibiotic activities of the enteric bacteria have been most intensively examined. Suggestive evidence that such inhibitory strains may be related to recovery from bacillary dysentery has been obtained.§ A previous paper from this laboratory [4] has described some rather striking general analogies between the situation in the intestinal tract and the ocular flora with respect to such bacterial inhibitors. The present report summarizes the survey findings relating such antibiotic-producing strains to the presence of clinical conjunctival inflammation in the groups examined previously.

MATERIALS AND METHODS

The subjects employed in this study were inmates of a New York state institution for the feebleminded.‖ They were of either sex, from 2 to 60 years of age, and resided in seven different cottages. The eyes were first examined clinically ¶ for the presence of conjunctival disease or other superficial infection. Culture material was then obtained from the lower conjunctival sac and lid margin (combined) of one eye, using broth-moistened swabs. These were then inoculated onto four culture plates in such a way that well-isolated colonies were usually obtained over one section of each plate. The basal medium employed was proteose #3 agar (Difco). Two plates were used with plain basal medium; one contained 5% human blood, and the other 5% human plasma, in addition. Discard bank blood was used for this purpose. After incubation for 48 hours, the plates were examined and typical colonies of each type noted were picked to fresh medium. Rough estimates of the numbers of each colony type were recorded. The replanted cultures were further examined by the Gram stain, for catalase activity,[13] mannitol fermentation, and coagulase

This investigation was aided by grants from the Eli Lilly & Company, and by the Snyder Ophthalmic Foundation.

From the Departments of Ophthalmology and Bacteriology, Columbia University College of Physicians and Surgeons, and the Institute of Ophthalmology, Presbyterian Hospital.

* References 1, 2, and 3.

† References 5, 6, and 7.

‡ References 8 and 9.

§ References 10, 11 and 12.

‖ Dr. Harry C. Storrs, Director of Letchworth Village, permitted the examinations to be made.

¶ Dr. L. von Sallmann and Dr. R. L. Wiggins assisted in making the clinical examinations.

production.[14] After the picking of sample strains, the entire plates were then spray-seeded with "susceptible" indicator cultures of ocular staphylococci, # albus variety #7-6, and aureus variety #15-1. They were then further incubated and examined for the presence of inhibitory colonies. Details of the methods and types of results obtained are recorded in a previous report.[4]

RESULTS

Clinical Findings.—A total of 280 patients were examined. One group was revisited several times at intervals of two to four weeks, so that a total of 359 observations were made. In 203 instances the conjunctivae appeared normal, and in 156 instances conjunctivitis of some type was present. A summary of the clinical diagnoses are shown in Table 1.

It may be seen that the predominant finding was catarrhal conjunctivitis, usually of a mild nature. It may also be pointed out that most of the instances of severe and

TABLE 1.—*Clinical Diagnoses of Subjects Studied Bacteriologically*

				Conjunctivitis (No. of Patients)				
				Catarrhal Conjunctivitis*				
Group (No. of Patients)	Sex	Age Range (Approx.), Yr.	Normal Eyes (No. of Patients)	Mild	Moderately Severe	Severe	Follicular Conjunctivitis	Angular Conjunctivitis
H (40)	M	15-25	23	13	0	0	3	1
Iota (40)	M	4-10	20	8	0	0	11	1
Q (40)	F	15-20	34	3	0	0	3	0
S (40)	F	5-12	21	9	0	0	10	0
Y (40)	F	4-10	34	3	0	0	2	1
Delta (40)	M	20-35	22	15	2	1	0	0
Theta (40)								
5th visit	M	25-50	13	19	3	3	0	2
6th visit	M	25-50	17	12	5	1	0	4
7th visit	M	25-50	19	15	2	2	0	2
280 patients								
359 observations			203	97	12	7	29	11
					116	156		

* For three patients included in this group the diagnosis was blepharoconjunctivitis.

moderately severe disease of this type appeared in Cottage Theta. Follicular conjunctivitis was absent in Cottage Theta and Cottage Delta.

In a few instances, in Cottage Theta only, clinical evidence was present for the diagnosis of healed trachoma. This disease had been found previously in this group by Thygeson and Braley.[16] Although conjunctival scrapings were not done routinely in the study reported here, on one occasion all the patients of this cottage were thus examined. Giemsa-stained preparations failed to reveal the presence of the inclusion bodies characteristic of trachoma in any case. It is felt, therefore, that this disease was not a factor in the current investigation.

The etiology of the "infections" observed is quite uncertain. Although conjunctivitis due to the Morax-Axenfeld diplobacillus (Moraxella lacunata) has also been observed previously in this institution,[16] organisms answering this description were found culturally on only four occasions, from three subjects in this series.

Although the currently preferred name for these organisms is Micrococcus pyogenes var. albus and M. pyogenes var. aureus,[15] we shall use the older designation of Staphylococcus albus and Staph. aureus for convenience.

Although conjunctival scrapings were not done routinely, and might have increased the positive findings for the Moraxella group, it is of interest that the relative scarcity of these organisms agrees well with recent findings by Gibson.[23] The data relating to the patients from whom Moraxella organisms were isolated are summarized in Table 2.

It is of some interest that in only one of these three patients was the disease diagnosed clinically as "angular conjunctivitis," the typical syndrome produced by infection with this agent.[17] A total of six patients in this study were given similar diagnoses, whereas another three were described as probably belonging to this group. Of these latter nine patients, only the one mentioned above revealed the diplobacilli culturally (as shown in Table 2). The other eight did not show any distinctive bacterial pattern in the ocular flora. It is necessary to point out that six of these nine patients with diagnoses of angular conjunctivitis were of the Mongoloid

TABLE 2.—*Patients from Whom "Diplobacilli" Were Isolated*

Patient	Cottage	Mongolism	Date	Clinical Diagnosis	Bacterial Flora (Cultures—Rough Colony Estimates)	Antibiotic Producers		
						HA*	SA	NA
1	Theta	—	11/27/50	Mild catarrhal conj.	10,000 "diplobacilli" 10,000 staphylococci	0	0	20
			12/11/50	Mild catarrhal conj.	5,000 "diplobacilli" 900 staphylococci	0	0	300
			1/ 8/51	Mild catarrhal conj.	2,000 staphylococci 50 "diphtheroids"	0	1	30
			1/22/51	Mild catarrhal conj.	850 staphylococci (mann.—; coag.—)† 15 streptococci	0	0	100
2	Theta	+	11/27/50	Angular conj.	10,000 staphylococci	1	0	100
			12/11/50	Severe angular conj.	50 Neisseria 10 staphylococci	0	1	50
			1/ 8/51	Severe angular conj.	10,000 "diplobacilli" 10,000 Neisseria	0	20	500
			1/22/51	Severe angular conj.	10,000 Neisseria 1,500 staphylococci (mann.—; coag.—)	0	10	10
3	Delta	—	2/ 6/51	Very mild chronic conj.	10,000 "diplobacilli" 1,000 staphylococci (mann.+; coag.—)	1	7	32

* HA are highly active antibiotic producers, as tested versus "Staphylococcus" 7-6 or 15-1; SA is slightly active, and NA is nonactive.
† Mannitol fermentation and coagulase production; — is negative; + is positive.

feebleminded type. Such persons seem especially susceptible to various infections, and they often have skin of very faulty texture and function.* In clear agreement with these reports, it was found here that 34 of the 44 (77%) Mongoloid patients revealed conjunctivitis clinically, while only 67 of the 215 (31%) non-Mongoloids showed such changes. Analysis of these data statistically reveals that such a difference would occur by chance less than once in 10,000 (i. e., $P = < 0.0001$). It is conceivable that the excoriation and erosion at the canthi seen in this group of patients causes the clinical resemblance to classical angular conjunctivitis.

Bacterial Findings.—The bacterial species found in the eyes of the subjects adequately studied are summarized in Table 3. Only those cultures showing moderate numbers of colonies of the recorded species are listed. Practically all of the cultures also revealed varying numbers of colonies of micrococci, usually of the Staph. albus group, as judged by detailed examinations of sample strains. It may

* References 18, 19, and 20.

be seen that no over-all striking differences in the flora between the normal and the conjunctivitis groups were observed. One possible exception to this statement was the finding of either Hemophilus or diplobacillus colonies in four of the conjunctivitis patients, while none were found in the control eyes. Analysis of the data for the presence of Staph. aureus strains in any concentration failed to reveal any significant differences between the two groups. Thus, on the basis of the data available, it was not possible to associate any given bacterial etiological agent with the conjunctivitides found here.

TABLE 3.—*Bacterial Flora of Normal and Conjunctivitis Eyes*

	Normal						Conjunctivitis					
Group	Staph. Aureus*	Strepto-cocci*	Hemo-philus*	Diph-theroids*	Diplo-bacilli	No "Patho-gens"	Staph. Aureus*	Strepto-cocci*	Hemo-philus*	Diph-theroids*	Diplo-bacilli	No "Patho-gens"
H	3†	0	0	0	0	20	5	0	1	1	0	10
Iota	3	0	0	0	0	17	4	1	0	(1)‡	0	15
Q	9	0	0	0	0	25	3	0	0	0	0	3
S	3	0	0	0	0	18	1	2	0	0	0	16
Y	6	4	0	1	0	23	2	(1)	0	0	0	4
Delta	1	0	0	1	0	20	5	(2)	0	0	1	12
Theta, 6th visit	0	3	0	1	0	13	3	2	1	1	1	14
Theta, 7th visit	0	4	0	1	0	14	3	5	0	4	0	9
Totals	25	11	0	4	0	150	26	10	2	6	2	83
				190						129		

* Only those cultures showing more than 50 colonies of these "species" are recorded here.
† Figures represent number of patients.
‡ Patients in ( ) showed two of the species indicated, usually with Staph. aureus.

TABLE 4.—*Patients Revealing Antibiotic Producers, in Relation to Clinical Conjunctivitis*

	Number of Patients with					
	Normal Conjunctivae			Conjunctivitis		
Cottage	HA*	SA†	Total No. Patients	HA	SA	Total No. Patients
Q	1	14	34	0	3	6
Y	2	6	33	0	3	7
S	1	9	21	0	9	19
Iota	0	6	20	0	4	20
H	2	11	23	1	8	17
Delta	3	17	2	1	14	18
Theta (last 3 visits)	4	28	48	18	19	69
Totals	13	91	201	20	60	156
	(6.5%)	(45.3%)		(12.8%)	(38.5%)	
	51.8%			51.3%		

* Patients revealing any highly active strains; some had both highly and slightly active organisms.
† Patients revealing any slightly active strains; all revealed nonactive strains in addition.

Findings Related to Antibiotic Activity.—The prevalence, in the normal and the "infected" groups, of persons with antibiotic-producing strains in their ocular flora is shown in Table 4.

There are no significant differences in the two groups with respect to the numbers of patients with slightly active strains. Although the differences observed with respect to highly active strains (12.8 versus 6.5% in "infected" and normal groups) would be due to chance alone in only 5 of 100 such series (i. e., the probability, P, equals 0.05), the sampling methods used here were not rigid enough to

allow reliance on any but very striking differences. It must also be pointed out that only in Cottage Theta was this "significantly" high rate of antibiotic-producing carriers associated with conjunctivitis.

The same type of observation also holds true with respect to estimations of the numbers of antibiotic-producing colonies observed on the plates from the normal versus the conjunctivitis series. These data are shown in Table 5.

Again, there is no significant difference between the normal and the "infected" eyes with respect to the numbers of slightly active strains, but the numbers of highly active colonies were more numerous in the conjunctivitis cultures than in the normal cultures. Statistical examination of this difference (0.16 and 0.50%) showed that it could be due to chance alone only 6 times in 1,000; i. e., *P* equals 0.006. Again, the sampling methods used prevent unqualified acceptance of the data, and, as in the previous case, it is only in Cottage Theta that this "significant" difference is apparent.

TABLE 5.—*Distribution of Antibiotic-Producing Colonies from Normal and Conjunctivitis Eyes*

	Number of Bacterial Colonies					
	Normal Eyes			Conjunctivitis		
Cottage	HA	SA	Total Observed	HA	SA	Total Observed
Q	1	63	1,186	0	11	156
Y	2	13	1,125	0	20	398
S	1	54	371	0	63	231
Iota	0	21	285	0	20	283
H	3	100	1,106	1	110	1,109
Delta	3	173	1,328	1	121	980
Theta (last 3 visits)	5	235	4,195	48	346	6,913
Totals	15 (0.16%)	659 (6.87%)	9,596	50 (0.50%)	691 (6.86%)	10,070
	7.03%			7.36%		

It seemed pertinent to inquire whether patients possessing Staph. aureus in their cultures were more likely than others to possess antibiotic-producing strains. Of the 255 patients on whom adequate information was available, 63 harbored typical Staph. aureus strains. In this group, 49% revealed antibiotic strains in their cultures. Of the 196 patients who were free of Staph. aureus, 54% revealed antibiotic producers. A breakdown of these data with respect to highly or slightly active strains still revealed no significant differences.

There also proved to be no relationship between the incidence of conjunctivitis in a given cottage and the concentrations of antibiotic-producing colonies and of persons carrying them. These data are shown in Table 6.

Although the cottages are arranged downward in order of increasing prevalence of conjunctivitis, the concentrations of inhibitory micro-organisms show no correlation whatsoever.

It is quite clear, however, that these inhibitory strains are not merely accidental passing transients or contaminants in the ocular flora of a given patient. On at least four occasions, Cottage Theta was revisited and follow-up observations made on the 40 patients under study there. It was common to find antibiotic producers

repeatedly from a single patient, often in approximately similar concentrations. A good example is shown in Table 7, which summarizes follow-up data from one patient on the last four visits.

That the active strains are part and parcel of the ocular flora itself is further supported by studies currently being done in this laboratory.[21] It has been found that much higher concentrations of antibiotic-producing micrococci may be found in the ocular flora when an exquisitely susceptible Corynebacterium is used as an indicator strain (C. pseudodiphtheriticum #A6485). In testing random samples

TABLE 6.—*Lack of Relationship Between Incidence of Conjunctivitis in a Given Cottage and the Prevalence of Antibiotic Producers*

Cottage	Incidence of Conjunctivitis	Incidence of Patients with Antibiotic Producers	Incidence of Antibiotic-Producing Colonies
Q	6/64 (9%)	18/40 (45%)	75/1,342 (6%)
Y	9/60 (15%)	11/40 (28%)	35/1,523 (2%)
Delta	17/104 (16%)	35/40 (88%)	298/2,308 (13%)
H	18/78 (23%)	22/40 (55%)	214/2,215 (10%)
S	20/70 (28%)	19/40 (48%)	118/602 (20%)
Theta	34/108 (31%)	69/117 (57%)*	632/11,108 (7%)*
Iota	20/50 (40%)	10/40 (25%)	41/568 (7%)

* These figures represent pools of the data obtained from 40 patients in Cottage Theta on three different visits two weeks apart.

TABLE 7.—*Serial Follow-Up of One Patient (25, M, W, Mongoloid)*

Date	Clinical Diagnosis	Number of Antibiotic-Producing Colonies		Total Colonies Examined	Bacterial Flora
		HA	SA		
11/27/50	Moderately severe chronic conj.	22 (6.0)*	3 (2.0, trace)	170	500 Staph. albus 400 Staph. aureus (?)
12/11/50	Mild chronic conj.	6 (6.0)	0	70	70 Staph. albus
1/ 8/51	Mild chronic conj.	5 (6.0)	0	25	600 Staph. albus 60 Staph. aureus (mann.+; coag.+)
1/22/51	Mild blepharoconj.	5 (7.0)	0	35	300 Staph. aureus (mann.+; coag.+) 200 Staph. albus

* ( ) represents size of inhibition zones of indicator strain, expressed in millimeters of width.

of 20 staphylococci from ocular cultures, it has been rather common to find 25 to 100% of the strains of a given patient to be inhibitory by means of antibiotic production. It is quite unlikely that these high concentrations of active micro-organisms merely represent chance contaminations.

In the studies reported here, using the staphylococcal strains as indicator organisms, no definite change in the antibiotic flora was found to correlate clearly with changing clinical status, when follow-up observations were made in Cottage Theta.

COMMENT

It is clear from these results that antibiotic-producing bacteria are found in the ocular flora of the eyes of patients with conjunctivitis, as well as from normal eyes. Unfortunately, quantitative aspects of the methods used are so comparatively inexact that inferences regarding the incidences of such active strains in the normal and

"conjunctivitis" groups must be made with caution. It may be stated, however, that no striking differences in the prevalence of such strains in the two groups were noted. There was a suggestion in Cottage Theta that patients with diseased conjunctivae were more likely than normal persons to reveal the presence of highly active antibiotic-producing strains. Such a finding, if confirmed by further data, would be similar to analogous observations in the intestinal tract. It was found by several investigators that Shigella- or Salmonella-infected patients were more likely than normals to reveal the presence of antibiotic-producing coliform strains in small samples.† In this case, of course, the bacterial pathogens (Shigella or Salmonella) are rather readily recognized and identified.

Although the findings presented above reveal that antibiotic organisms are to be found in diseased ocular flora, they shed little light on the possible role of such strains in resistance mechanisms. It must be pointed out, however, that these conjunctivitis patients almost all had a chronic inflammatory disease and that the etiological agents were indeterminable. It is planned that more pointed studies be carried out, in which the changes of the bacterial flora are examined (with respect to antibiotic activities) during the course of acute conjunctivitis, where a bacterial agent may be more clearly implicated. It is hoped that changes in the characteristics of the flora may be followed and that any antibiotic-producing strains isolated may be tested for activity against the infecting agent.

The Mongoloid feebleminded proved to have a significantly higher incidence of conjunctivitis, as compared with the non-Mongoloids. These patients in a previous study revealed higher susceptibility to Shigella infections [10] as well. Their generally low resistance to infections is well recognized, and the findings here with respect to superficial ocular disease adds a further example of this phenomenon.

The general patterns of the bacterial species found in these eyes are in very good agreement with a recent study by Gibson, in Queensland, Australia.[23] In his series of normal eyes the principal species found were staphylococci and "diphtheroids." Of the latter, about one-fifth were coagulase-positive. His conjunctivitis series failed to reveal the diplobacillus of Morax-Axenfeld in any instance. Most of these latter patients revealed staphylococci principally (115/248), while streptococci and pneumococci were not uncommon (24/248 and 16/248, respectively). Also, he states that "the diplobacillus of Morax-Axenfeld was not found in any normal eyes, or in any eye with an appearance of angular conjunctivitis." The latter observation supports the observations made here in the Mongoloid patients.

SUMMARY

1. Institutional feebleminded patients with normal eyes, as well as those with chronic conjunctivitis of various clinical types, revealed antibiotic-producing organisms in the ocular flora. No striking differences in the incidence of antibiotic strains were found in the two groups.

2. No correlation was found between the prevalence of antibiotic-producing strains and the incidence of conjunctivitis in the various cottage groups examined.

3. Mongoloids were more likely than the undifferentiated feebleminded to show clinical evidence of conjunctivitis.

† References 10, 12, and 22.

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VISUAL EFFECTS OF PINK GLASSES, GREEN WINDSHIELDS, AND GLARE UNDER NIGHT-DRIVING CONDITIONS

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THERE can be no doubt that automobile travel at night is dangerous when the driver wears colored glasses.[1] There has been, however, no agreement about how much of any color, or what combinations of color, can be considered safe for general public use in glasses and windshields. The tinted glass used is not dark enough to change the color of traffic signals significantly, although there have been complaints about low brightness and poor color rendition of outdoor drive-in movies. The chief objection to the use of tinted glass is the possibility of accident due to reduced visual efficiency at night.

Particularly unfortunate is the popular selection of pink for the glasses and aquamarine green for the windshields. While pure red and pure green filters may be quite transparent, in combination they are opaque. How much loss of vision at night might be expected from pink glasses and green windshields? Does the alleged relief from glare and fatigue at night compensate for the reduced visual efficiency?

When a person first walks into a dark movie theater, he can see the screen very well, but there is poor visibility of the seats until dark adaptation has occurred. Night driving is a similar visual task, except that dark adaptation can never take place. Cone dark adaptation begins at twilight, 1 to 0.1 mL (the millilambert is a unit of light intensity equal to 0.929 foot-candle, or f.-c.) and continues to about 0.01 mL, which is full moonlight. This degree of dark adaptation is accomplished in a few seconds and increases absolute light perception about tenfold. Rod dark adaptation begins at about 0.1 mL, which is deep twilight, and becomes complete at 0.001 mL, which is quarter-moonlight.

The intensity of illumination from a pair of automobile headlights turned on bright is approximately 100 mL at 9 ft. (2.7 meters) ahead, 10 mL at 28 ft. (8.4 meters) (quite adequate for prolonged reading), 1.0 mL at 90 ft. (27 meters), and 0.01 mL at 900 ft. (270 meters). The driver's eyes become adapted to the average reflectance of this light from objects on and about the road. Adaptation varies with changes of gaze, but the process is so slow that it should be considered constant until a passing car produces temporary full light adaptation. The light reflected from a painted white dividing line might be 70%, from concrete 50%, and from black top 10%.

Therefore, for night driving, the eyes remain in the photopic state, with adaptation for an intensity of at least 0.1 mL. At this level, the seats in a dark movie theater would remain forever black against black, just as the objects at a distance or in the shadow appear on the road.

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TABLE 1.—*Derivation of the Luminosity Curves*

Wave Length	Energy Auto Headlight	Luminosity Curve 0.1 F.-L.	Luminosity No Glass (1 × 2)	Transmission, Per Cent Crown	Luminosity Through Crown (3 × 4)	Transmission, Per Cent Yellow	Luminosity Through Yellow (3 × 6)	Transmission, Per Cent Pink	Luminosity Through Pink (3 × 8)	Transmittance Green	Luminosity Through Green (3 × 10)	Transmittance Green + Pink (8 × 10)	Luminosity Green + Pink (3 × 12)
	1	2	3	4	5	6	7	8	9	10	11	12	13
350	0.000	0.00	0.00	0.830	0.0	0.000	0.00	0.00	0.00	0.000	0.00	0.000	0.00
60	0.000	0.00	0.00	0.890	0.0	0.000	0.00	0.15	0.00	0.357	0.00	0.054	0.00
70	0.000	0.00	0.00	0.908	0.0	0.000	0.00	0.35	0.00	0.495	0.00	0.173	0.00
80	0.001	0.01	0.00	0.912	0.0	0.000	0.00	0.60	0.00	0.465	0.00	0.279	0.00
90	0.003	0.02	0.00	0.917	0.0	0.000	0.00	0.70	0.00	0.553	0.00	0.387	0.00
400	0.009	0.08	0.00	0.920	0.0	0.000	0.00	0.76	0.00	0.612	0.00	0.465	0.00
10	0.017	0.20	0.00	0.920	0.0	0.000	0.00	0.76	0.00	0.618	0.00	0.470	0.00
20	0.027	0.70	0.02	0.920	0.0	0.000	0.00	0.73	0.01	0.618	0.01	0.451	0.01
30	0.039	1.60	0.06	0.920	0.1	0.000	0.00	0.70	0.04	0.620	0.04	0.504	0.03
40	0.063	3.00	0.15	0.920	0.2	0.000	0.00	0.65	0.11	0.621	0.10	0.404	0.06
450	0.070	4.91	0.34	0.920	0.4	0.000	0.00	0.64	0.24	0.631	0.22	0.404	0.14
60	0.090	7.76	0.70	0.920	0.7	0.010	0.00	0.65	0.46	0.642	0.45	0.417	0.28
70	0.114	11.78	1.34	0.920	1.2	0.020	0.03	0.66	0.90	0.649	0.87	0.427	0.60
80	0.142	17.96	2.55	0.920	2.3	0.057	0.14	0.67	1.71	0.652	1.66	0.437	1.13
90	0.171	27.46	4.69	0.920	4.3	0.200	0.94	0.69	3.23	0.655	3.07	0.452	2.12
500	0.201	41.70	8.38	0.920	7.7	0.680	5.72	0.70	5.86	0.655	5.49	0.458	3.83
10	0.231	61.75	14.26	0.920	13.0	0.810	11.50	0.71	10.12	0.656	9.36	0.466	6.64
20	0.262	79.22	20.75	0.920	19.2	0.862	17.92	0.72	14.94	0.654	13.57	0.471	9.77
30	0.293	91.08	26.69	0.920	24.2	0.889	23.74	0.72	19.22	0.651	17.35	0.469	12.51
40	0.325	98.00	31.85	0.920	29.2	0.907	28.84	0.73	23.25	0.648	20.58	0.473	15.07
550	0.357	100.00	35.70	0.920	32.8	0.918	32.77	0.73	26.06	0.642	22.92	0.469	16.73
60	0.389	97.33	37.86	0.920	34.8	0.923	34.98	0.73	27.64	0.639	24.19	0.459	17.37
70	0.421	90.80	38.22	0.920	35.2	0.925	35.42	0.74	28.28	0.632	24.18	0.468	17.90
80	0.453	80.28	36.36	0.920	33.5	0.925	33.67	0.76	27.63	0.625	22.75	0.475	17.26
90	0.485	67.39	32.68	0.920	30.0	0.923	30.08	0.77	25.16	0.618	20.19	0.476	15.54
600	0.517	54.43	28.14	0.920	25.9	0.921	25.88	0.77	21.66	0.610	17.18	0.470	13.25
10	0.549	41.71	22.89	0.920	21.0	0.919	20.95	0.76	17.39	0.600	13.75	0.456	10.48
20	0.581	29.53	17.16	0.920	15.8	0.911	15.66	0.75	12.87	0.590	10.12	0.443	7.60
30	0.613	19.74	12.10	0.920	11.1	0.908	10.98	0.74	8.95	0.572	5.94	0.423	5.12
40	0.645	12.24	7.89	0.920	7.2	0.905	7.06	0.74	5.84	0.564	4.45	0.418	3.30
650	0.677	7.11	4.81	0.920	4.4	0.902	4.33	0.77	3.70	0.550	2.65	0.423	2.06
60	0.709	3.90	2.77	0.920	2.6	0.900	2.52	0.78	2.16	0.534	1.48	0.416	1.15
70	0.741	2.06	1.48	0.920	1.4	0.897	1.34	0.80	1.20	0.514	0.75	0.411	0.58
80	0.773	1.00	0.77	0.920	0.7	0.893	0.71	0.85	0.65	0.494	0.38	0.420	0.32
90	0.805	0.52	0.40	0.920	0.4	0.890	0.35	0.87	0.37	0.475	0.21	0.413	0.16
700	0.837	0.27	0.26	0.920	0.2	0.889	0.18	0.89	0.25	0.464	0.12	0.413	0.09
10	0.869	0.13	0.11	0.920	0.1	0.887	0.08	0.90	0.09	0.456	0.05	0.411	0.04
20	0.899	0.07	0.05	0.920	0.0	0.885	0.05	0.90	0.00	0.434	0.02	0.391	0.02
30	0.926	0.03	0.02	0.920	0.0	0.883	0.02	0.90	0.00	0.420	0.01	0.378	0.00
40	0.950	0.02	0.00	0.920	0.0	0.880	0.01	0.91	0.00	0.400	0.00	0.364	0.00
750	0.971	0.00	0.00	0.920	0.0	0.879	0.00	0.91	0.00	0.385	0.00	0.350	0.00
60	0.988	0.00	0.00	0.920	0.0	0.878	0.00	0.91	0.00	0.367	0.00	0.334	0.00
70	1.000	0.00	0.00	0.920	0.0	0.874	0.00	0.91	0.00	0.348	0.00	0.316	0.00
			391.4		359.6		345.9		290.00		244.09		180.16
			(100%)		(92%)		(88.4%)		(74%)		(62.4%)		(46.5%)

As the driver studies the road at the distance limits of the headlights, he constantly tests his visual thresholds. Objects come into view, attract attention, and are finally identified, as the automobile rapidly approaches. Under threshold conditions, an image may form on the retina 50 times and be so weak that only 25 attention responses follow. Any decrement in illumination or visual efficiency during high-speed night driving could delay reaction enough to result in serious accident.

Modern windshields were made green because large areas of glass let in too much heat from the sun. A green filter cuts out the red and infrared rays which carry heat. For purposes of night driving this windshield color becomes the worst possible selection, because automobile headlight is unbalanced. Almost two-thirds of headlight energy is concentrated in the red end of the spectrum, and only one-third is in the range to which a green windshield is most transparent.

Exact figures on visual efficiency through tinted glass in night driving can be obtained from basic data now in the literature. The headlight illuminant consists of the percentage energy for each wave length specified in Table 1, Column 1.[2] The relative sensitivity of the human fovea to each of these wave lengths is given in Column 2. Because of the Purkinje shift during dark adaptation, this relative luminosity was determined under night-driving conditions at 0.1 foot-lambert[3] (0.107 mL). By multiplying the items for each wave length in Columns 1 and 2, a new luminosity curve for night driving was derived, Column 3. This typical luminosity curve is illustrated as the upper dotted line in Chart 1, labeled *3*, the column number in Table 1.

To determine the relative luminosity for vision through clear crown glass, multiply the 100% luminosity Column 3 item for item by Column 4, which is the crown transmission column. The result, Column 5, is the night-driving luminosity curve for clear glass. In Chart 1, it appears as a dashed line just inside the dotted one. The sum of the transmission Column 5 is 92% of the sum of Column 3 and represents the average relative transmission of crown glass. This 8% loss is almost entirely by reflection from the two surfaces; so thickness is not very important.

Similar calculations for the lightest shade of yellow, which has enjoyed some popularity as a night-driving glass, appear in Columns 6 and 7, Table 1. The yellow luminosity curve in Chart 1 is a solid line very near that of crown glass and has a blue cut-off at about 490 μ. Under night-driving conditions, the average transmission of the yellow filter is 88.4%.

The second shade of pink glass varies slightly by brand, but is typified by Column 9, Table 1, and by Curve *9*, Chart 1. The average transmission of this glass under these conditions is 74.0%. The transmissions in Table 1 for crown, light-yellow, and the second shade of pink glasses were obtained from the National Bureau of Standards Circular 471, 1948. The transmission of green windshield glass was measured for both brands in Washington University radiation laboratory. The darker one was selected here, but the differences are insignificant.

The night-driving luminosity curve for green windshield glass appears in Column 11, Table 1, and in the dashed line *11*, Chart 1. The average transmission under these conditions is 62.4%. Multiplying each item of transmission of pink glass, Column 8, by that for green windshield, Column 10, gives the combined transmission, Column 12. This column multiplied by the luminosity, Column 3, gives the luminosity through both pink glasses and the green windshield, Column 13, Table

1, and the solid line *13*, in Chart 1. This combination has an average transmission of 46.5% and is a dark filter. The occurrence of this combination at night on our highways is not at all rare.

I have repeated these calculations for dark adaptation to 0.01 foot-lambert, which corresponds to full moonlight with headlights turned off. The average luminosity through crown glass remained the same, but decreased to 82.8% in yellow glass, and to 71.8% in pink glass. Therefore, tinted glass becomes even more dangerous at night when the headlights are turned down or when the intensity is diminished by mud or mechanical defect.

There are individual differences in the luminosity curve which may make slight differences in the visual effects of tinted glass My calculations have shown these to be unimportant except in the case of true color blindness. In this common condition,

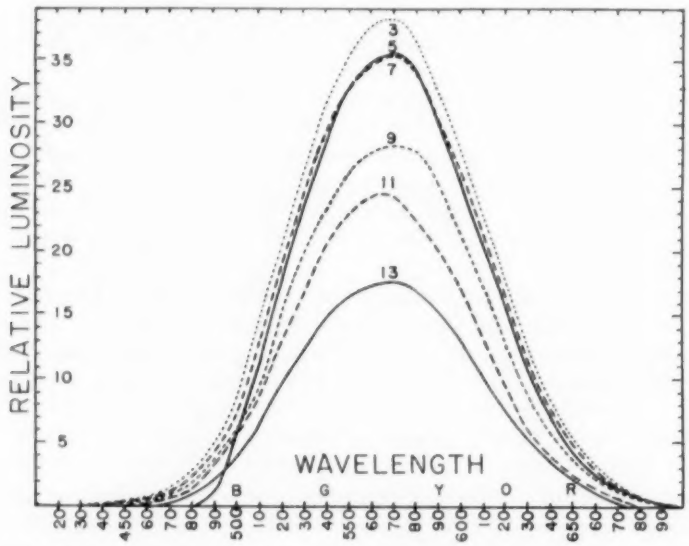

Chart 1.—Relative luminosity curves under night-driving conditions with vision through no glass (dotted line, *3*), crown (dashed line *5*), light-yellow (solid line *7*), second-shade pink (dashed line *9*), green windshield glass (dashed line *11*), and the combination of pink glass and green windshield (solid line *13*). The numbers refer to the columns of origin in Table 1.

there is not only a shift in the luminosity curve along the spectrum, but, more important, a reduced average luminosity. In deuteranopia, the commonest type of color blindness, I find the luminosity for night driving through crown glass to be reduced from 92 to 90.8%. Luminosity through yellow night-driving glasses changes from 88.4 to 82.9%. Even the slightest tinted glass adds to the visual problem at night for color-blind persons.

RELATION OF VISUAL EFFICIENCY TO LIGHT INTENSITY

The effect on visual efficiency of reducing light intensity by filters has been thoroughly studied. Part of the enormous literature is summarized in Charts 2 and 3, in which the number on each curve refers to the visual sensitivity scale of the columns on the left edge of each chart and to the number column in Table 2.

Suppose that the eyes are adapted to the reflectance of concrete pavement at a distance of 90 ft. from the car, an intensity of about 0.1 mL through colorless glass. To determine the effect on visual acuity * of the yellow, pink, green, and combined pink and green filters under consideration here, locate Curve *1*, Chart 2. From the 0.1 mL position on the light intensity scale, raise a perpendicular to Curve *1*. Measure this distance with dividers and lay it on the visual acuity scale, Column 1 at the left. The visual acuity through clear glass under night-driving through yellow night-driving glass to 0.088 mL, the perpendicular distance to Curve *1* corresponds to a visual acuity of 20/34.

Thus, pink glass reduces visual acuity to 20/40, and green windshield to 20/46, and the combination of the two to 20/60. These figures are tabulated with other measurements of visual efficiency in Table 2.

Although all these studies were made in the absence of glare, the presence of glare during night driving would not invalidate them. There is no evidence that

TABLE 2.—*Effect of Tinted Glass on Various Functions of Vision*

Visual Thresholds	Figure	No. in Fig.	Crown	Yellow	Pink	Green	Pink-Green
Adaptation level, mL	..	..	0.1	0.088	0.074	0.062	0.046
Visual acuity	2	1	20/32	20/34	20/40	20/46	20/60
Stereopsis [7]	2	2	12.2″	12.8″	13.6″	14.4″	15.8″
Discrimination of angular velocity, in sec. arc/sec.[8]	2	3	120″	135″	145″	170″	205″
Discrimination span of instant counting [9]	2	4	4.3	3.3	2.0	0.6	0
Resolving power [4]	3	5	9.8″	28″	40″	42″	43″
Discrimination of simultaneous contrast [5]	3	6	27%	30%	33%	37%	43%
Discrimination of intensity change, green [10]	3	7	69%	65%	70%	73%	79%
Discrimination of direction of line target [11]	3	8	4.1″	4.9″	5.5″	6.2″	7.1″

* Troland, L. T., cited by Evans.[3]

color filters improve visual acuity under ordinary night driving conditions with glare from oncoming automobiles. They may improve comfort and reduce fatigue. To confirm the effect of colored filters on visual acuity during night driving in the presence of glare, I devised the following experiment:

The subject was seated at one end of a large room with dull black walls. Behind him was low diffuse illumination of about 0.1 f.-c. to make handling of apparatus easier. A Clason visual-acuity meter was used in which the voltage was reduced by a variable transformer until the test letters on the screen 20 ft. (6 meters) away reflected 0.1 f.-c. of light. To measure this amount of light with a photographic light meter, I measured the light at the screen and at the projector with the voltage at 135, then reduced the voltage until the light at the projector became proportional to that desired from the screen.

Five degrees (approximately 2 ft., or 60 cm.) to one side of the screen was placed a 6-volt, 108-watt, ribbon filament lamp operated at 5.5 volts to reproduce the color temperature of automobile headlight. Fitted with a + 20.00 D. lens, this lamp projected a 6-ft. blur circle of light about the subject's head at an eye-position intensity of 2.5 f.-c.

* References 4, 5, and 6.

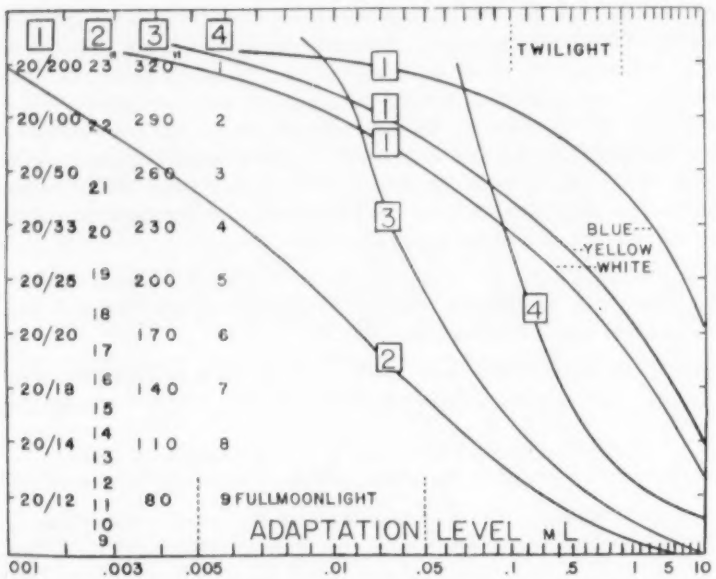

Chart 2.—Effect of the intensity of the adaptation level of light, in millilamberts, on (*1*) visual acuity, (*2*) stereopsis, (*3*) discrimination of angular velocity in seconds arc per second, and (*4*) span of instant counting.

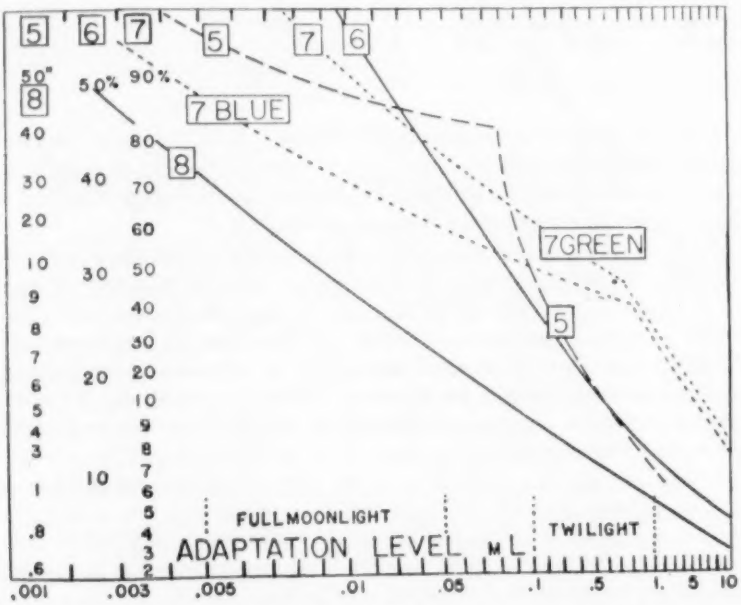

Chart 3.—Effect of the intensity of the adaptation level of light, in millilamberts, on (*5*) resolving power, (*6*) discrimination of simultaneous contrast, (*7*) discrimination of intensity change, (*8*) discrimination of direction of a line target.

The visual acuity was first tested without glare by gradual enlargement of test type until reading became possible. Then, the glare was turned on, the patient being cautioned not to look directly at it. Then binocular visual acuity was tested several times until the subject became somewhat adjusted to the glare. The initial loss of acuity from glare was even more striking than indicated by Chart 4. Vision was tested through yellow night driving glasses, the second shade of pink glasses, a piece of green automobile windshield, and, finally, through combined pink glasses and green windshield.

The results are presented in Chart 4. The thick solid line indicates the changes in visual acuity predicted by calculation. The thin solid lines represent measured visual acuity under simulated night driving conditions through tinted glass in subjects who complained of glare sensitivity. The dashed lines represent similar tests

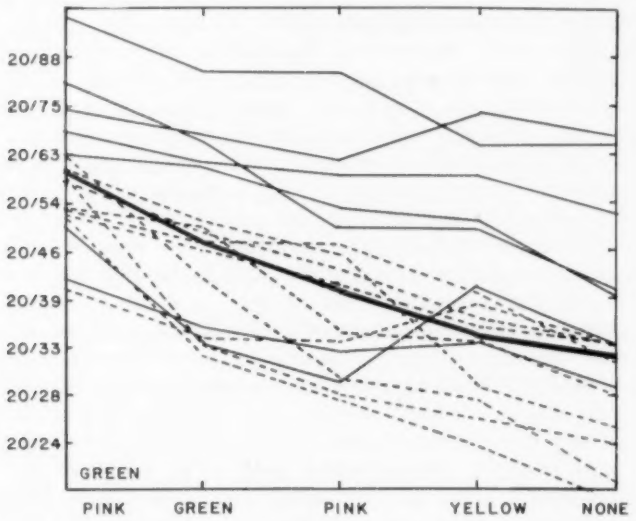

Chart 4.—Effect of tinted glass on visual acuity of subjects having normal daylight vision, taken under simulated night-driving conditions against glare. The solid lines represent subjects who complained of discomfort from glare, while the broken lines represent those without complaint.

of subjects having no complaint from glare. All subjects had normal visual acuity by ordinary tests. Those in the habit of wearing glasses used them in the experiment. The loss of visual acuity from glare was roughly proportional to the complaint.

That light-yellow night-driving glasses would reduce the visual acuity surprised me, because the luminosity curve closely resembles that for crown glass. The pink glass used in the experiment was much darker than the yellow, without proportional loss of visual acuity. Three subjects had equal visual acuity through green windshield and pink glasses. One of these habitually wore dark green glasses indoors and out, day and night. I suspect that persons in the habit of wearing tinted glasses learn to select certain color bundles in the fovea and to suppress others.

This experimental situation differed from actual road conditions in various ways, but the gradual increase in size of stimuli until recognized was quite appro-

priate. I tried using 20/40 test type and gradually increasing the illumination until it became legible, but the results were variable. In the real situation, visual efficiency is decreased by object movement, dust, rain, jolting car movements, fatigue, boredom, alcoholism, and various distractions. I believe these would only add to the significance of my figures.

EFFECT OF GLARE ON VISUAL ACUITY

Partial night blindness and excessively slow light-dark adaptation occur in otherwise normal persons. Since night driving is a photopic vision problem, one might mistakenly assume that such changes would be unimportant. Peripheral retina being anatomically adapted for night vision, the presence or absence of tinted glass should make no difference. There are differences, however, which include the sensitivity of peripheral retina to glare or stray light within the eye.

Complaints from glare in night driving are commoner in persons who have colloidal changes in the ocular media, either from disease or from old age. In such eyes incoming light rays are scattered by dispersion, in the familia, Tyndall phenomenon. A similar dispersion can be observed in night driving with a slightly muddy windshield. As long as there are no oncoming cars, visibility is excellent. However, when oncoming lights strike the windshield, it becomes white and opaque.

In dispersion, there is a theoretical reason for the use of light yellow night driving glasses. Since dispersion is inversely proportional to the fourth power of the wave length, about 16 times more blue light rays are scattered in the ocular media than red. A yellow filter would remove the blue light and prevent some of the confusion. Practically, automobile headlight is red in color and contains so little blue that a light-yellow filter probably makes little difference.

There is another source of stray light in the eye which would also be reduced by a yellow filter. Because of chromatic aberration, normal eyes are a diopter or more myopic to blue. Additional night myopia is present in advanced levels of dark adaptation. Again, there is not much blue light involved in night driving, and there has been no evidence of improvement of vision by correcting chromatic aberration, although this work should be repeated under night driving conditions.

Albers and Sheard [12] made rough estimates of the effect on visual acuity of glaring colored light. Although the physical intensity was equal, the blue glare source proved relatively more objectionable. Wright [13] found that blue light seemed to exhaust some substance in the retina which required 175 seconds of time to be replaced, while with the same quantity of green or red light but 100 seconds was required. An equal amount of yellow light exhausted its substance, with a recovery time of only 20 seconds.

Under threshold visual conditions, there are many intraretinal and extraretinal reflex and perceptive phenomena which are at present poorly understood. Our eyes provide us with pictures which make sense, but what we think we see is often much different from the physical retinal image. There are individual variations in suppression of blur or color fringes, and in the weight for judgment conferred upon certain qualities of the retinal stimulus. A person with excellent visual acuity may have poor contrast discrimination. Such phenomena may explain the widespread subjective improvement in comfort and in visual acuity reported from the use of light-yellow night-driving glasses. Perhaps some function of vision may be improved, but not visual acuity, because under my simulated night driving conditions the average visual acuity dropped from 20/36 to 20/42.

SUMMARY AND CONCLUSIONS

The use of tinted glass in automobiles is dangerous because of decreased visual efficiency at night. Under night-driving conditions the visual acuity is 20/32 on Snellen type through colorless glass. Light-yellow night-driving glasses change this to 20/34. The second shade of pink glass reduces visual acuity to 20/40, while the green windshield glass reduces it further to 20/46. The combination of pink glasses and green windshield is particularly bad, resulting in a visual acuity of 20/60.

Even more damning is the effect of tinted glass on resolving power during night driving. The threshold is decreased from 10 to 42 seconds arc by the green windshield alone. A pair of objects would appear separate at 100 ft. through a clear windshield, which would appear single through a green windshield until the distance had decreased to 25 ft. Other factors of vision, like stereoscopic acuity, instant counting span, or discrimination of angular velocity, simultaneous contrast, intensity change, or direction of a line target, are likewise defective in night-driving conditions with tinted glass.

Green windshield glass should be in a separate layer, to be moved aside for night driving. Persons with defective vision, including color blindness of the common type, should be advised to add auxiliary headlights to their automobiles and avoid any type of tinted glass for night driving. Since glare is inversely proportional to the area of the source, automobile headlights should be designed larger in area.

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STUDY OF THE COMPENSATION-MAXIMUM TEST ON AQUEOUS VEINS

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KLEINERT and Grün [1] and Kleinert [2] observed in eyes suffering from different types of glaucoma a retrograde influx of blood from episcleral veins into aqueous veins during compression of the eyeball. The dynamometer pressure necessary to produce this phenomenon was termed compensation maximum. In normal-pressure eyes compression of the eyeball up to 150 gm. usually would not produce this backflow phenomenon, while in glaucomatous eyes in many instances a pressure of 100 gm. or less sufficed to force back blood into the aqueous veins. The authors discussed extensively this phenomenon, which, in their opinion, proved helpful for clinical clarification of suspected cases of glaucoma.

Kleinert [2] offered three explanations of the compensation-maximum phenomenon. In the first he quoted Lindner as assuming that during compression of the eyeball more than the usual amount of aqueous is expressed and leaves the inner eye through deep outlets and that this increased outflow produces a suction-like effect on the superficially visible aqueous veins, causing influx of blood into the latter. If, however, the superficial pathways are sufficient to permit the forcibly increased outflow, Lindner expects that no backflow of blood will be observed.

Another explanation offered by Kleinert was that during compression of the eyeball an increase in pressure inside the episcleral veins produces the backflow phenomenon in the aqueous veins. This effect is favored by the increased pressure in epibulbar veins during the increasing phase of the pressure in glaucomatous eyes. Kleinert raised a third point by assuming that during compression of the eyeball the elimination of aqueous from the canal might be interfered with and therefore the entrance of blood into the aqueous veins might be facilitated.

While Kleinert's original readings were performed with the ophthalmodynamometer of Müller, he later employed finger compression, reserving the dynamometer for doubtful cases (personal communication, May 29, 1952).

The compensation-maximum test was studied in the ophthalmology department of the Cincinnati General Hospital according to the procedure described by Kleinert and Grün. Although our observations have been far fewer than the 400 reported by these investigators, certain trends are apparent.

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METHOD

The aqueous veins were first located and their position, type, contents, and any particular characteristics noted. Using the ophthalmodynamometer of Bailliart, pressure (up to a maximum of 150 gm., unless interrupted by discomfort of the patient) was gradually applied by a second observer through the lids toward the center of rotation of the globe. Each 10-gm. increment of pressure was indicated to the first observer, who continually watched the vessel system. In each instance the point of pressure was so placed that the outflow of the recipient vessel was not embarrassed. The lids were manually retracted when this was necessary for biomicroscopic visualization. In more recent studies, as noted later, pressure was sustained at 150 gm. for variously timed intervals. Progressive changes back to the original appearance of the outflow system were timed after pressure release. Tonometry was never done earlier than one hour before these tests. On the other hand, none of the data are based on tonometric values following compression tests obtained on the same day, as these values are significantly lowered by the manipulations. Further studies were abandoned on a few subjects who found ocular compression uncomfortable. In no case was topical anesthesia used. A single complication was a bulbar subconjunctival hemorrhage at the site of compression, which resolved in a few days. All the glaucomatous eyes in this study were of the chronic normal-angle noncongestive type in various stages of progression, the diagnosis having been established by the combined evidences of tonometry, ophthalmoscopy, gonioscopy, field studies, and provocative tests.

OBSERVATIONS

In a preliminary study conducted between the end of 1951 and the summer of 1952, a series of 61 eyes, 46 with normal pressure and 16 glaucomatous, were subjected to the compensation-maximum test (Table 1). On repeated readings, 30 of the normal eyes had a high compensation maximum; i. e., the aqueous veins remained clear even at pressures of 150 gm. Ten of the normal-pressure eyes showed low compensation-maximum readings; i. e., blood entered the aqueous veins with pressures less than 150 gm. Six eyes were inconsistent in their response, showing high or low compensation-maximum readings at various times, whether tested on different days or in a series of observations performed on the same day, allowing up to 60 minutes between tests. If the test was repeated within a few minutes, in many eyes there was a tendency toward lower compensation-maximum readings. When we performed 121 compensation-maximum tests on 46 normal-pressure eyes, only 54 tests showed Kleinert's normal high value of 150 gm. Forty tests gave widely scattered readings, 17 of which fell between 140 and 100 gm. and 23 below 90 gm.; among the latter were tests resulting in blood influx at pressures of 40 gm. (three times), 30 gm. (once), and 20 gm. (twice).

Twenty-seven observations were omitted from the group of normal-high compensation maximum tests because of difficulty in classification: For technical reasons the pressure of 150 gm. was not reached, and therefore it was uncertain whether an aqueous vein, clear to, say, 140-gm. pressure, would have remained so could 150-gm. pressure have been applied.

Another difficulty was a fleeting blood influx appearing in a few aqueous veins at a certain level of external pressure and disappearing as this was raised. These observations were not excluded from the group of high compensation maxima. Among the inconsistent normal-pressure eyes in our study there was one showing high compensation maximum readings on repeated tests over a four-month period and a low compensation-maximum reading only once. It is interesting that persons

with two normal-pressure eyes may show a high compensation-maximum reading in one and a low reading in the fellow eye. To date, none of the eyes in the normotensive group with low readings have shown any sign of glaucoma.

Of 15 glaucomatous eyes, only 2 eyes of the same person had a high compensation-maximum value of 150 gm. 12 hours after the last pilocarpine administration; 9 showed filling of the aqueous vein with blood at readings varying from 30 to 140 gm., and 5 responded inconsistently to different examinations. No statistically significant relation was found between intraocular-pressure values and compensation maximum-test values.

Summarizing 35 observations on glaucomatous eyes, we found only 2 eyes with a consistently normal compensation maximum; in 14 tests values between 140 and 100 gm. were found, and in 19 tests the values were below 90 gm. Among the last were tests resulting in blood influx at a pressure of 40 gm. (three times) and 30 gm. (twice). For two eyes with Schiøtz readings between 31 and 45 mm. Hg "normal" compensation-maximum values occurred at least temporarily.

By medically controlling the tension in two glaucomatous eyes, a change from low to high compensation-maximum values was observed; in five other glaucomatous eyes the compensation maximum was unchanged, regardless of a medically induced drop in tension.

TABLE 1.—*Compensation-Maximum Test in Normal and Glaucomatous Eyes*

	Total Eyes	High Compensation Maximum	Low Compensation Maximum	Inconsistent Readings
Normal	46	30	10	6
Glaucomatous	16	2	9	5

While the essence of the compensation-maximum test is the observation of the aqueous vein during vigorous compression of the eyeball, the compression test on the recipient vessel * is concerned with the study of the aqueous vein during a gentle blocking of its flow. In normal-pressure eyes both aqueous influx and blood influx may be observed during the compression of the recipient vessel. In glaucomatous eyes blood influx is the rule. The results of the influx phenomenon and the compensation-maximum tests, performed on the same aqueous veins, were compared in a series of 13 normal and 12 glaucomatous eyes. In aqueous veins with high compensation-maximum readings the aqueous influx prevailed in normal-pressure eyes at a ratio of 4:1, and the blood influx in glaucomatous eyes, at a ratio of 3:1. In both normal and glaucomatous eyes, aqueous veins with low compensation-maximum readings showed a distribution of five blood-influx to three aqueous-influx phenomena.

While the original compensation-maximum test consisted of observation of the aqueous vein during rapidly increasing external pressure, one of us (J. L. S.) suggested that the test might be elucidated by observing the aqueous vein during sustained ocular compression at 150 gm. for 20 seconds and noting its characteristics following pressure release. On the basis of a limited number of observations, the characteristics after pressure release seemed to have little significance, and the sustained compression showed too inadequate a consistency to be of value, as

* References 3, 4, 5, 6, 7, 8, and 9.

illustrated in Table 2. Furthermore, of 11 persons subjected to the sustained compression test on both eyes, only 4 showed identical responses, whereas 7 demonstrated differing responses.

The next question was whether the results of the compensation-maximum test and the sustained compression test corresponded to each other, and if so how closely. This comparison was made on 40 eyes (Table 3).

Examination of the data reveals the variability of response to these tests not only in different patients having the same clinical diagnosis, but also in the two eyes of the same patient. The following case shows how manifold the responses of an individual eye may be.

TABLE 2.—*Response of Aqueous Veins to Sustained 150-Gm. Pressure for Twenty Seconds*

	Total Eyes	Aqueous Vein Remained Clear	Aqueous Vein Filled with Blood	Changing During Observation	Varying from One Test to Another
Normal	25	11	8	1	5
Glaucomatous	16	4	8	0	4

TABLE 3.—*Comparison of Compensation Maximum and Sustained Ocular Compression in Normal and Glaucomatous Eyes*

Response during sustained compression	Total Eyes	High Compensation Maximum	Low Compensation Maximum	Variable Response
Aqueous vein clear				
Normal	11	10	0	1
Glaucomatous	4	2	0	2
Aqueous vein filled with blood				
Normal	8	2	6	0
Glaucomatous	7	0	6	1
Variable response				
Normal	5	1	1	3
Glaucomatous	5	1	1	3
Total	40	16	14	10

A 45-year-old patient with unilateral chronic simple glaucoma showed two aqueous veins in her glaucomatous eye. During compression of the recipient vessel, the large aqueous vein showed aqueous influx; the smaller aqueous vein, blood influx. Both these aqueous veins showed high compensation-maximum readings. With sustained pressure the aqueous vein with the blood-influx phenomenon showed blood filling and the aqueous vein with the aqueous-influx phenomenon remained clear. On another occasion, when the tension had risen, the eye then being less well controlled by pilocarpine, both aqueous veins showed blood influx during compression of the recipient vessel and during sustained ocular compression, and low compensation-maximum values. This patient was examined gonioscopically during compression of the eyeball. While 130-gm. pressure was applied to the bulbus for 30 seconds no blood was seen in Schlemm's zone. When 150-gm. pressure was applied for 20 seconds to the eyeball of another patient suffering from simple glaucoma, blood entered the canal; and with release of pressure blood suddenly rushed from the canal. The same result was obtained on repetition of this test. On the same day, an aqueous vein of this eye showed the aqueous-influx phenomenon but a low compensation maximum, and blood filled the aqueous vein during compression.

COMMENT

Kleinert's statement that high compensation maxima are characteristic of normal-pressure eyes and low compensation maxima of glaucomatous eyes was generally confirmed in our rather small series. However, the high percentage of normal eyes showing high compensation-maximum values (92%), as reported by Kleinert, was not found in our group of normal eyes, of which only two-thirds showed high values.

The majority of the glaucomatous eyes showed low compensation-maximum values.

Our findings are in contrast to those of Kleinert, who stated that on repetition of the compensation-maximum test during the same observation period practically identical figures were found. Six out of 61 eyes showed inconsistent readings, and persons with normal-pressure eyes may show high values in one eye and low values in the fellow eye.

If only 60% of the definitely proved cases of chronic simple glaucoma showed low compensation-maximum readings, it is hardly likely that those eyes showing compensation-maximum readings of less than 100 gm. should "almost always" later show at least transient tension elevations, as Kleinert reported.

On the basis of our observations, the following relationship may be derived: In a given aqueous vein, the combination of aqueous influx, a compensation maximum of 150 gm., and persistent clearness of the vessel during sustained compression is found oftener in normal than in glaucomatous eyes. However, the combination of the reverse of each of these conditions not infrequently occurs in normal eyes and hence does not necessarily indicate glaucoma. Similarly, while low resistance to outflow is the rule in normal-pressure eyes during tonography, high resistance may also be found.†

The fact that in one eye different aqueous veins may show different responses to (*a*) compression of the recipient vessel, (*b*) the compensation-maximum test, and (*c*) sustained pressure suggests that the results of these tests are not characteristic of the clinical diagnosis of this particular eye, not even of its intraocular pressure, but only of pressure differences between aqueous and blood at the point where they meet in the recipient vessel.

The response of a given aqueous vein to mechanical manipulation and, as previously shown,‡ to pharmacological factors is usually a constant and reproducible characteristic of the individual vessel, dependent upon the anatomical configuration, the available connections of that vessel, and the hydrodynamic and physiologic factors governing the relative powers of the aqueous and blood streams at their junction.[8] De Vries [14] found that the aqueous influx occurs in aqueous veins with rapidly streaming contents and the blood-influx phenomenon in aqueous veins with slow current. It is very probable that these differences are directly related to anatomical differences, as described by Ashton § and by Thomassen and Bakken [17]; large aqueous veins deriving directly from wide canal outlets must have a more vigorous fluid output than those deriving from small outlets or passing through a complicated vascular meshwork. It is very probable that these differences will also

† References 10, 18, and 19.

‡ References 11, 12, and 13.

§ References 15 and 16.

influence the result of the compensation-maximum test. The explanations of this test as offered by Lindner and by Kleinert may well be reconciled with the above-outlined anatomical viewpoint. In this connection the observations by Moses and

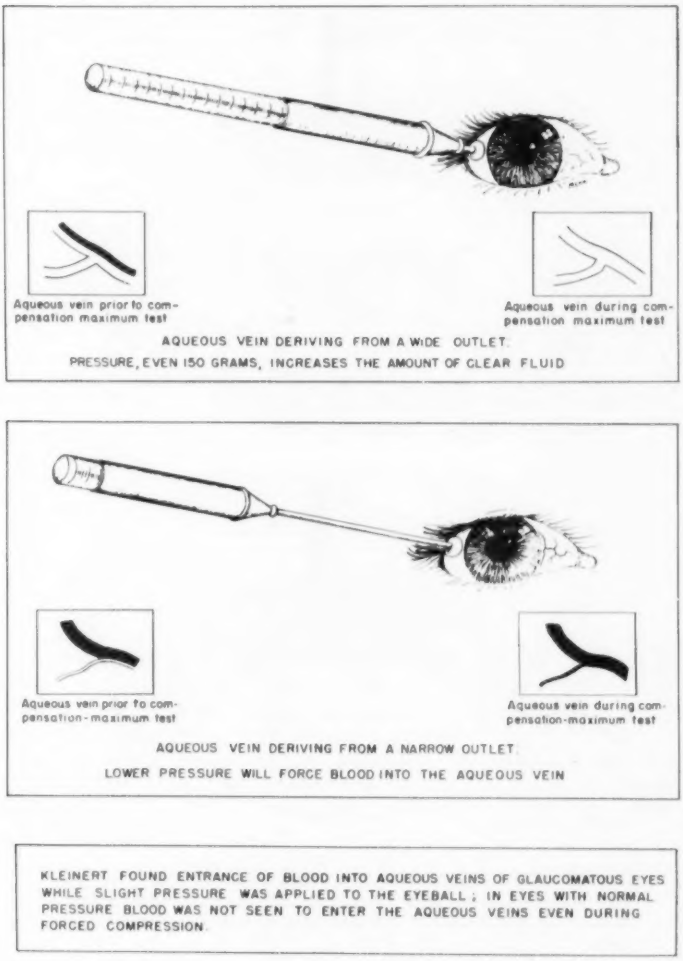
COMPENSATION-MAXIMUM TEST (KLEINERT, 1951)
Aqueous vein prior to compensation maximum test
Aqueous vein during compensation maximum test
AQUEOUS VEIN DERIVING FROM A WIDE OUTLET.
PRESSURE, EVEN 150 GRAMS, INCREASES THE AMOUNT OF CLEAR FLUID
Aqueous vein prior to compensation-maximum test
Aqueous vein during compensation-maximum test
AQUEOUS VEIN DERIVING FROM A NARROW OUTLET.
LOWER PRESSURE WILL FORCE BLOOD INTO THE AQUEOUS VEIN
KLEINERT FOUND ENTRANCE OF BLOOD INTO AQUEOUS VEINS OF GLAUCOMATOUS EYES WHILE SLIGHT PRESSURE WAS APPLIED TO THE EYEBALL; IN EYES WITH NORMAL PRESSURE BLOOD WAS NOT SEEN TO ENTER THE AQUEOUS VEINS EVEN DURING FORCED COMPRESSION.

Figure 1

Bruno [10] should be mentioned. They found a direct linear relationship between tonometric pressure and rate of flow of aqueous up to pressures of about 47 mm. Hg and beyond this a marked increase in the rate of flow in some eyes. This might indicate that some eyes have available additional channels of outflow which remain collapsed unless needed during periods of increased intraocular pressure. It is

understandable that aqueous veins deriving from wide outlets may provide for additional outflow irrespective of whether under natural, pathologic, or experimental conditions. Although too few observations have been made to date to be convincing,

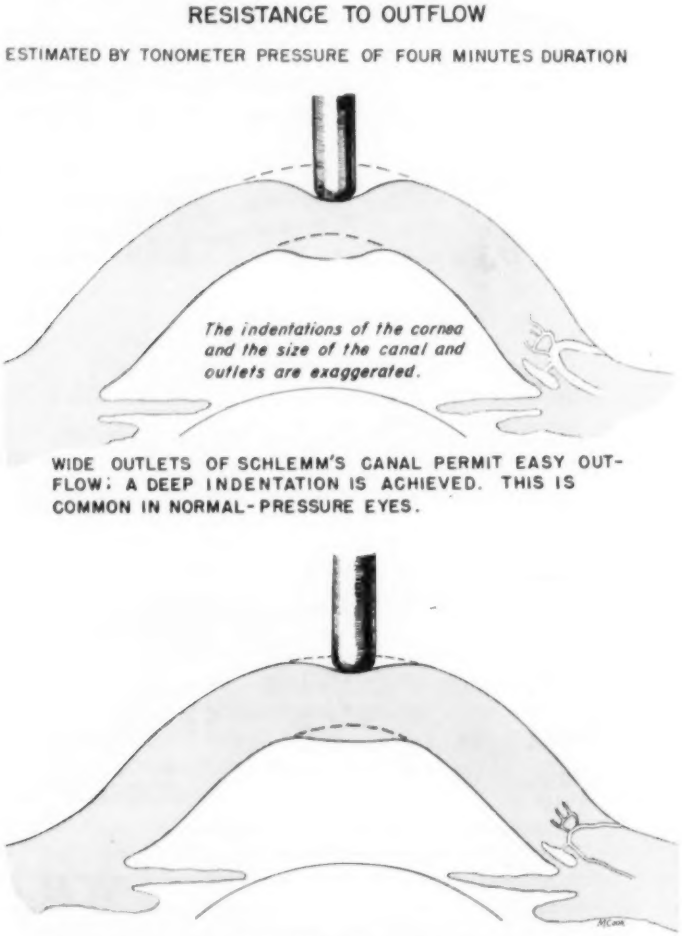
RESISTANCE TO OUTFLOW
ESTIMATED BY TONOMETER PRESSURE OF FOUR MINUTES DURATION
The indentations of the cornea and the size of the canal and outlets are exaggerated.
WIDE OUTLETS OF SCHLEMM'S CANAL PERMIT EASY OUTFLOW; A DEEP INDENTATION IS ACHIEVED. THIS IS COMMON IN NORMAL-PRESSURE EYES.
NARROW OUTLETS RESTRICT THE AQUEOUS OUTFLOW; THE INDENTATION REMAINS SHALLOW. THIS IS TYPICAL FOR GLAUCOMATOUS EYES BUT ALSO OCCURS IN NORMAL-PRESSURE EYES.

Figure 2

the results point in the same direction as former studies,‖ which indicated that a vigorous output of aqueous humor is encountered more frequently in aqueous veins of normal-pressure eyes, while a less powerful elimination of aqueous may occur in both normal and glaucomatous eyes.

‖ References 5, 6, 8, and 9.

In brief, tests performed on single aqueous veins study particular outflow channels and not the eye as a functional whole.

SUMMARY

While the compensation-maximum test more frequently yields high values in normotensive eyes and low values in glaucomatous eyes, in our experience it has not been sufficiently correlated or characteristic to be of value in clinical differentiation of these two groups. Rather, it reflects the outflow facility of the particular aqueous vein in question.

Dr. Edward J. Glaser participated in the early part of this work, and Mrs. June MacLean gave technical assistance.

5 W. 4th St. (Dr. Ascher).

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RECESSION UNDER TENON'S CAPSULE

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WHEN JAMESON first presented his technique of recession, he primarily described a method of suturing a muscle ending directly to the sclera.* This gave more uniform results than simple tenotomy. He advised that after the incision of the conjunctiva, the "capsule" be buttonholed about 3 mm. from the belly of the muscle in the region of the equator. A strabismus hook was inserted into the buttonhole and slipped under the muscle. A second buttonhole was made on the other side of the muscle at a corresponding distance. The muscle then was undermined, and diverging "capsular" incisions were made, which were carried forward to meet the muscle at the outlines of its insertion. When the muscle was tenotomized, a "capsular" flap overlying the muscle was completed and allowed to retract along with the muscle.

There have been many modifications of the Jameson original procedure. For example, the angle and placement of the conjunctival incision have been modified, and the type of scleral stitch and suture materials used have varied; however, the principle of incising the "capsule" over the muscle insertion and allowing it to retract seems to have persisted.

It is the purpose of this report to present a technique of handling Tenon's capsule which has been used at the University of Oregon Medical School since 1949. It has been directed toward maintaining the normal anatomy of Tenon's capsule and check ligaments without detracting from the efficacy of the surgical procedure.

In the literature there is no unanimity of opinion concerning the anatomical subdivisions of Tenon's capsule; therefore, some definition of terms is necessary. Anterior to the insertion of the rectus muscle the sclera is covered by thin reticular episcleral tissue which underlies and is loosely adherent to the conjunctiva. It seems to be the forward extension of Tenon's capsule. Posterior to the insertions of the rectus muscles the tissues covering the globe divide into more definite layers. A deep layer of reticular tissue covers the sclera between the rectus muscles and splits over and under these muscles to form capsules. This tissue lying under Tenon's capsule is spoken of as the "intermuscular membrane," although occasionally it may be too delicate to be considered a membrane.[3] From the region of the insertion of the muscles to behind the equator, Tenon's capsule is well defined as

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* References 1 and 2.

a layer of yellowish areolar tissue, which can be separated readily from the overlying bulbar conjunctiva and the underlying muscle capsules and intermuscular membrane. Posteriorly Tenon's capsule fades into the tissues of the orbit and fornices. In fact, many authors consider the portion of Tenon's capsule which covers the globe posteriorly as merely a condensation of the fatty tissues of the orbit.

The anatomy of the check ligaments also is disputed. In the fornices there are definite connective tissue reflections from Tenon's capsule to the orbital walls which "check" extreme rotations of the globe, but at surgery it is found that the strands which connect Tenon's capsule with the underlying muscle capsules can be separated easily. The insertions of the rectus muscles, therefore, can be transplanted, leaving undisturbed the reflections of Tenon's capsule to the orbital walls.

Tenon's capsule forms the natural bed for the bulbar conjunctiva. Where a defect is made in the capsule, cicatricial tissue is apt to bind the conjunctiva to the sclera and restricts its natural sliding motion with rotations of the globe. When Tenon's capsule is incised parallel to the insertion of the muscle, as in standard techniques of recession, the sclera is bared and permits the conjunctival incision to adhere to the globe. Also, when Tenon's capsule is allowed to retract with a tenotomized muscle, it may form a scar tissue mass deep in the fornix. This is particularly prone to happen if there is any appreciable postoperative inflammation. Then the rolled-up, retracted Tenon's capsule may form such a dense cicatrix in the fornix that the results of recession surgery may be nullified.

Some reasons why it is desirable to maintain the normal relationship of Tenon's capsule to the globe have been discussed. We first attempted to reach this objective by incising the conjunctiva and Tenon's capsule between the limbus and the muscle insertion. Then Tenon's capsule and conjunctiva were lifted as a single layer, under which the intermuscular membrane could be buttonholed and a hook slipped under the muscle. In this technique, the muscle was recessed or resected under Tenon's capsule; however, a large incision was required to give adequate exposure for a recession to the equator. Also, patients noticed considerable irritation from the sutures when they were close to the limbus. Finally, exposed cicatricial reactions were prone to occur with the conjunctival incision so far forward. It was decided, therefore, to make a conjunctival incision well behind the muscle insertion and parallel to the limbus; then to incise Tenon's capsule at right angles to the conjunctival incision, that is, parallel to the muscle. This would permit the splitting and retraction of Tenon's capsule and thereby expose the intermuscular membrane and the muscle in its capsule. After the muscle surgery was completed, Tenon's capsule could be closed separately from the conjunctiva. The conjunctiva would not be in contact with the stump of the muscle or with the bare sclera at any point. Furthermore, the conjunctival incision would lie in the fornix, where the sutures would produce little irritation or discomfort to the patient. The technique has been used since 1949 in more than 500 operations on the medial and lateral rectus muscles. It has been our experience that the splitting of Tenon's capsule parallel to the muscles permits excellent exposure and produces less postoperative reaction and irritation to the patient than the standard Jameson technique. With lateral rectus surgery, the inferior oblique insertion is simpler to locate and is more easily avoided. Also, complications, such as retraction of the caruncle and granuloma

formation, are almost unknown. (Reoperation has revealed striking differences between cases in which this technique was used and those in which Tenon's capsule was incised parallel to the limbus and allowed to retract. In cases where Tenon's capsule was split over the muscle at the time of surgery and then resutured, a minimum of subconjunctival scar tissue reaction has been found, whereas some degree of cicatricial reaction under the conjunctiva has been evident in almost all other cases.)

It might be assumed that if a muscle was recessed or resected leaving Tenon's capsule intact, the amount of correction would be lessened as compared with recessing or resecting Tenon's capsule along with the muscle. A review of cases

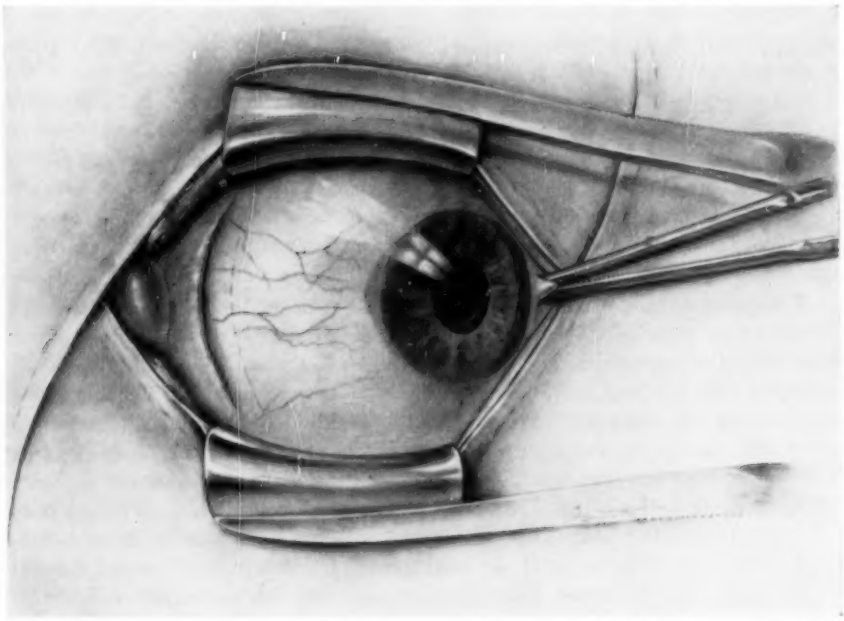

Fig. 1.—A self-adjusting drape is placed under the speculum blades and covers the lids. Just anterior to the plica the conjunctiva is incised without cutting the underlying Tenon's capsule.

in which recessions were performed on both medial rectus muscles in the same patient but at different times and with both techniques of handling Tenon's capsule revealed no significant difference in the amount of correction obtained with the later technique. In fact, the results were more consistent, due to lessened cicatricial reaction. The technique of splitting and retracting Tenon's capsule is applicable both to recession and to resection operations, as well as most tucking or partial tenotomy procedures. As recession of the medial rectus muscle is the commonest strabismus operation, it will be described.

The preoperative preparation of the eye for recession surgery does not differ from other operative procedures. We prefer to trim the lashes so that the patient

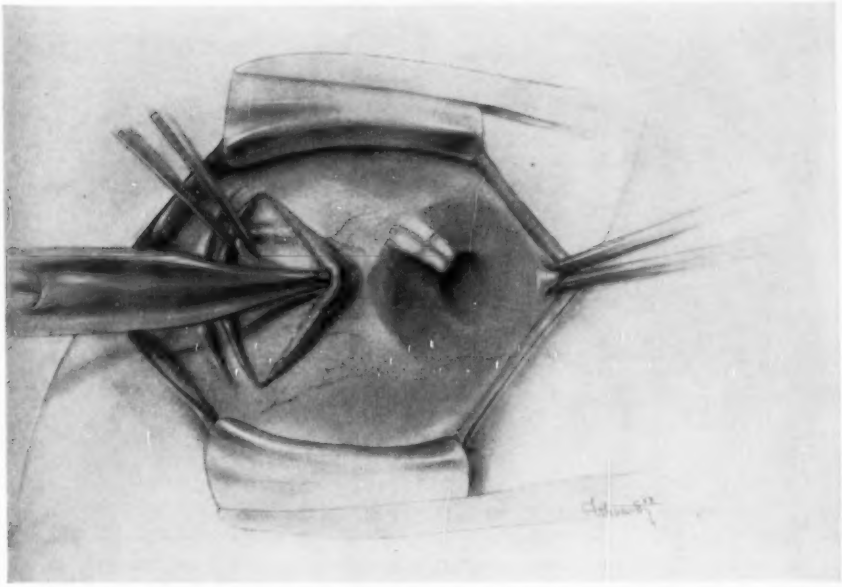

Fig. 2.—The conjunctiva is stripped back. Tenon's capsule is exposed over the muscle. Tenon's capsule only is grasped to avoid tearing of the fragile conjunctiva.

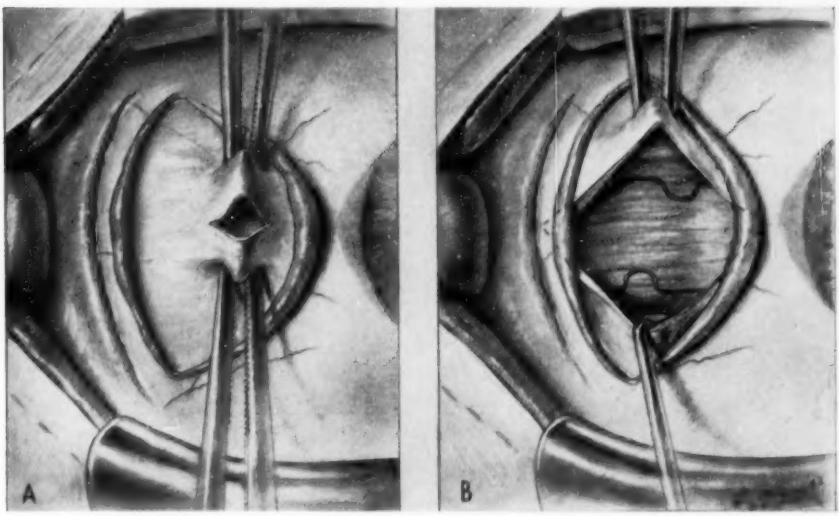

Fig. 3.—(*A*) Tenon's capsule is incised just anterior to the muscle insertion but at right angles to the conjunctival incision. (*B*) The incision is extended (parallel to the muscle). The muscle and intermuscular membrane are exposed by retraction of Tenon's capsule.

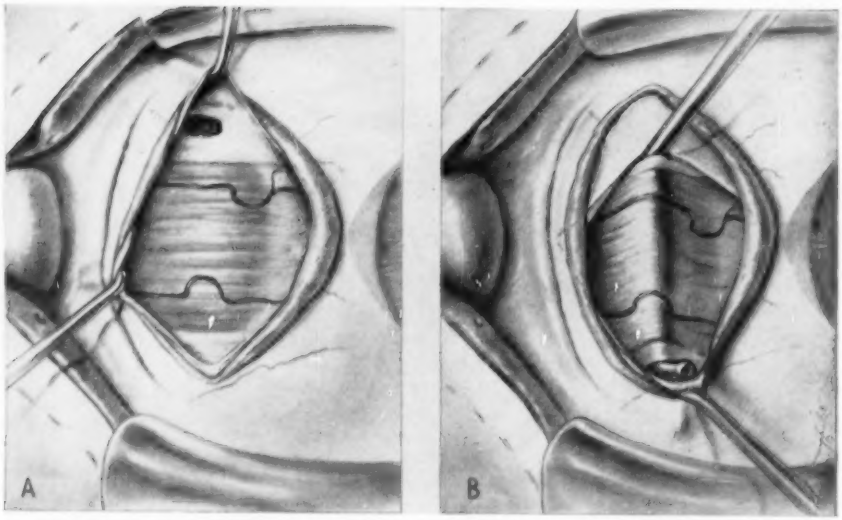

Fig. 4.—(*A*) The intermuscular membrane is buttonholed at least 2 mm. from the muscle belly and in the region of the equator. Incisions made closer to the muscle may result in retraction of the capsule and baring of the muscle fibers. (*B*) A hook is slipped under the muscle to facilitate buttonholing on opposite side.

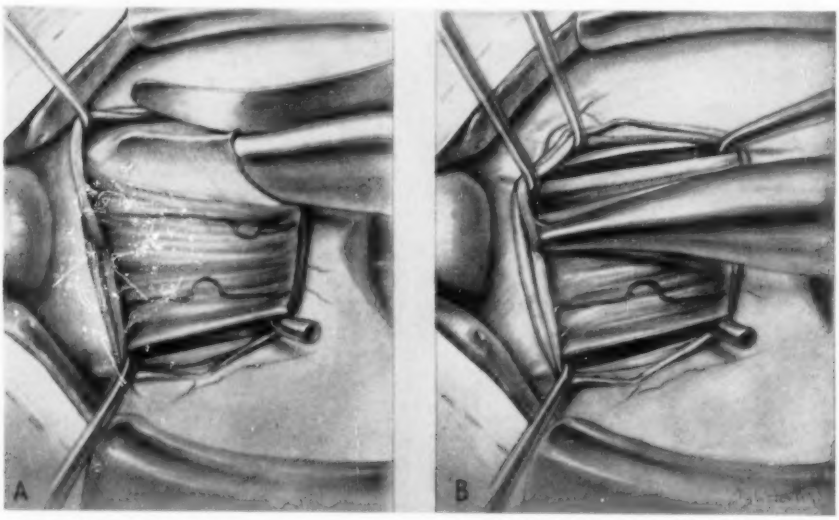

Fig. 5.—(*A*) Incisions in intermuscular membrane are extended parallel to the muscle. (*B*) Reflections of muscle capsule to Tenon's capsule are separated.

will not be bothered by the lashes sticking together after surgery. Also, there is less danger of contamination of the sutures by lashes protruding into the operating field. The lids and conjunctiva are cleansed in the usual manner. In this institution, a collyrium of silver proteinate is used to stain and coagulate the mucus. When this stained tissue is washed from the conjunctival sac with saline, we are reasonably certain of working in a clean field. Contamination of the sutures by the lash line and skin seems an important factor contributing to postoperative reaction; therefore, a spacial drape has been designed to cover the lash line. The opening is a slit created by overlapping two ellipses of cloth in such a manner that the edges fit the lid margin under the speculum. The slit will open or close in accordance with the degree of separation of the speculum blades.

With the eye abducted, the conjunctiva is picked up with forceps and incised about 1.5 mm. in front of the plica (Fig. 1). If the incision is made too deep in the fornix, some contraction of the fornix may occur. Also, the greater distance from the incision to the insertion necessitates a longer conjunctival incision. The length of incision depends upon the anatomy of the individual eye. Tenon's capsule is now grasped with forceps in the middle of the incision, and, using the blunt end of tenotomy scissors or a closed forceps tip, the conjunctiva is stripped back for 1 to 2 mm. on the plica side but at least 3 to 4 mm. on the corneal side (Fig. 2). This mobilization of the conjunctiva permits its separate closure at the conclusion of the operation. It is important to grasp Tenon's capsule with the forceps, rather than the conjunctiva, which is fragile and tears easily.

In the next step a small scissors incision is made in Tenon's capsule at right angles to the limbus (Fig. 3 *A*). This incision is made anterior to the insertion; otherwise the capsular vessels are apt to be cut and brisk bleeding ensue. One now sees sclera with a thin episcleral covering over the anterior perforating vessels. The blade of the scissors then enters the buttonhole, and Tenon's capsule is incised back toward the plica, usually with a single clean cut over the center of the muscle (Fig. 3 *B*). Tenon's capsule can now be retracted, exposing the muscle and its capsule with the intact intermuscular membrane visible on each side. No attempt is made to free Tenon's capsule from the plica and caruncle, as they may lead to disturbance of the check ligaments and to cicatrix formation with later retraction of the caruncle.

The next step is to buttonhole the intermuscular membrane so that bare sclera is exposed. This is done in the region of the equator and at least 2 or 3 mm. from the muscle belly (Fig. 4 *A*). If the buttonhole is placed closer, the intermuscular membrane, which splits to form the muscle capsule, will retract and bare the muscle fibers. (It is desirable to keep the thin capsule intact to minimize bleeding and postoperative adhesions.) A muscle hook is then slipped through the intermuscular membrane and is passed completely under the muscle to a point 2 or 3 mm. on the other side of the muscle, where again the intermuscular membrane is buttonholed (Fig. 4 *B*).

With the globe abducted, the fine reflections of the muscle capsule to Tenon's can be separated easily. Incisions are made in the intermuscular membrane parallel to the muscle up to its insertion (Fig. 5). In the splitting and retraction of Tenon's capsule the insertion of the muscle is already exposed cleanly, and further dissection around the insertion is rarely necessary. With the hook under the

muscle belly, the surgeon is ready to place the sutures. Muscle clamps have not been used at the University of Oregon Medical School during the last five-year period; instead, the muscle hook is slid back 3 to 4 mm. from the insertion and the muscle is so tented that the sutures can be placed close to the insertion.

We have preferred the lock stitch, as taught by C. S. O'Brien (Fig. 6). It has advantages over a mattress suture in that the muscle can be pulled snugly against the sclera before a knot is tied. Also, there is little danger of the suture pulling out of the muscle during the operation. By using a suture at each side of the muscle, it is possible to spread the muscle evenly over the sclera where it is to be sutured. In recent years we have used 0000 mild chromic absorbable surgical (gut) sutures

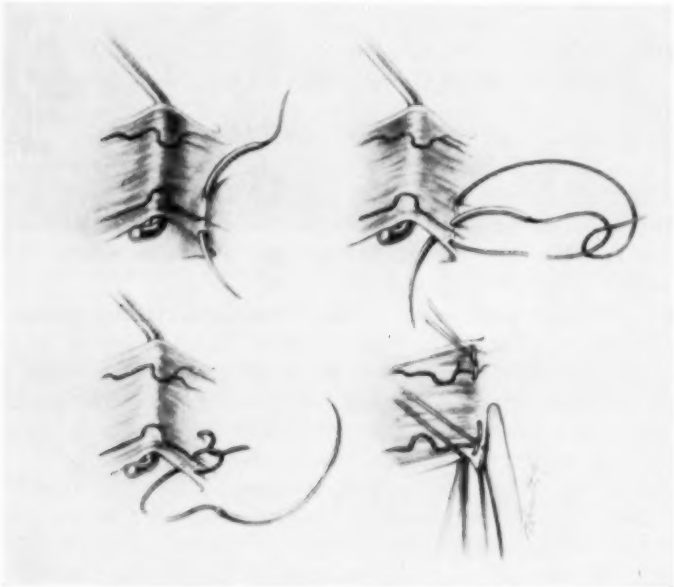

Fig. 6.—O'Brien lock stitches in the tendon are so placed that they ligate the anterior ciliary vessels. Blades of tenotomy scissors are held flat against the globe to recess all possible tendon.

on small atraumatic cutting needles. The stitches can be so placed that the anterior ciliary vessels are ligated. Bleeding after the tenotomy thereby is minimized.

As only bare sclera remains when the muscle has been recessed within its intact capsule, no disturbance or roughening of the sclera is necessary. In determining the placement of the scleral bites, the original insertion of the muscle is used as a guide. The two sutures are placed in the sclera parallel to it (Fig. 7 *A*). Calipers can be used to be certain that the two bites in the sclera are equally distant from the old insertion. In the tying of the scleral stitches, care is taken to invert the ends of the muscle tendon toward the sclera. This is best done by placing the knot on the scleral side rather than on top of the muscle (Fig. 7 *B*).

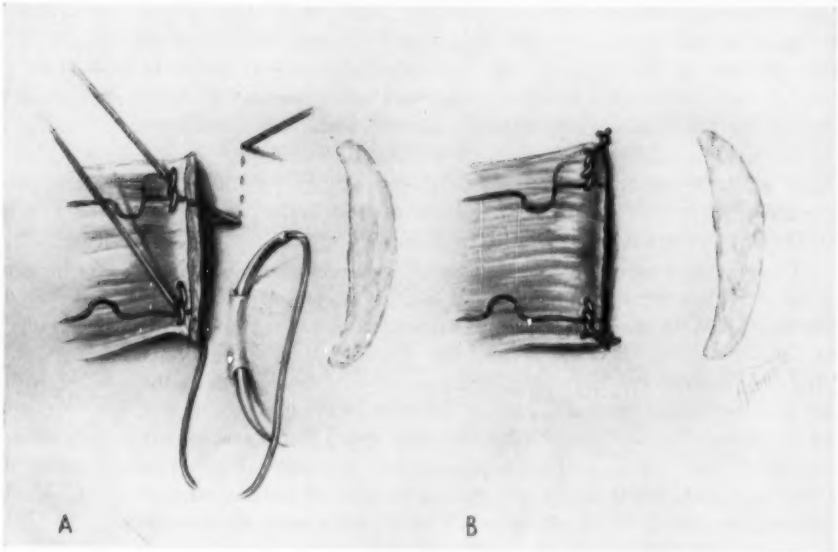

Fig. 7.—(*A*) The scleral stitches are placed parallel to and behind the extremities of the old insertion. (*B*) Knots are tied on the sclera so as to invert the cut end of the tendon against the sclera.

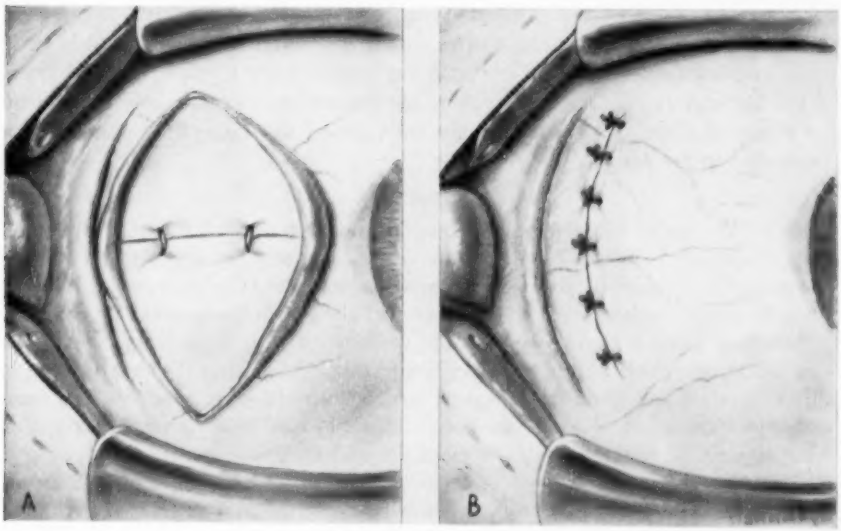

Fig. 8.—(*A*) Tenon's capsule is closed with fine absorbable sutures. The knot is placed under the capsule. (*B*) Conjunctiva is closed separately with fine absorbable sutures.

After the scleral sutures have been tied, Tenon's capsule is closed with one or two bites of 000000 mild chromic gut (Fig. 8*A*). It is important that these bites be placed where the stitch has no opportunity to protrude through the conjunctival incision; that is, the stitch or stitches should be placed so that it is covered by a flap of conjunctiva. The reaction seems less when the knot is tied on the inside, toward the muscle, rather than on the outside, under the conjunctiva.

The conjunctiva is closed separately with either interrupted or continuous sutures (Fig. 8*B*). If the line of conjunctival closure is kept free of Tenon's capsule and is mobile, sutures will seldom tear or cause distress to the patient. We prefer to use 000000 mild chromic gut and thereby eliminate the need for removing sutures.

Postoperative care for recession operations conducted by the above technique is simple. Only the eye operated on is bandaged. The bandage is usually removed the day following surgery. In many instances the eye not operated on is occluded to force the use of the eye operated on. No irrigating solutions or ointments are used postoperatively. Generally, the patients are discharged to their home with the sole instruction to keep the lids clean with a dampened cloth. For this purpose, we advocate a heavily piled, clean washcloth which has been laundered many times and is lint-free. Sterile cotton or gauze is not necessary. Furthermore, strands of cotton are apt to catch on the lash line, on sutures, or in the conjunctival sac. Most children are permitted to return to school within a week after surgery.

If the technique of splitting and retraction of Tenon's capsule is applied to a resection operation, no change in the incision is necessary, but care is taken to advance the intermuscular membrane along with the muscle, thereby retaining more nearly normal anatomy. The sutures attaching the new muscle ending to the old insertion are covered by both Tenon's capsule and conjunctiva.

In operations on the lateral rectus muscle, the initial conjunctival incision is made midway between the equator and the fornix. Care is taken to avoid ducts of the lacrimal gland. The incision through the conjunctiva is largely covered by the lids, and, therefore, the sutures are not exposed and cause little postoperative discomfort to the patient.

SUMMARY

With the usual techniques of recession and resection of the rectus muscle, Tenon's capsule is incised parallel to the limbus and at right angles to the muscle insertion. Most surgeons also incise Tenon's capsule alongside the muscle; thereby, a flap of Tenon's capsule is recessed or resected along with the muscle. This method of handling Tenon's capsule permits the conjunctiva to come in contact with the bare sclera, to which it readily becomes adherent. Also, after Tenon's capsule is allowed to retract, it tends to roll up and form a cicatricial mass which may decrease mobility of the globe. Finally, the check ligaments may be seriously altered. For these reasons, it seems desirable in resection and recession operations to preserve the normal relationships of Tenon's capsule. Also, it is desirable to place the conjunctival incision well away from the limbus to minimize postoperative irritation and cicatrization.

The above objectives have been achieved by placing the conjunctival incision behind the muscle insertion and parallel to the limbus. The conjunctiva is stripped from Tenon's capsule by blunt dissection. Then, Tenon's capsule is incised in front of the muscle insertion but at right angles to the limbus. The incision is then

extended over the muscle. When the edges of Tenon's capsule are retracted, the muscle is exposed within its capsule. The intermuscular membrane also is well exposed so that it can be buttonholed and incised cleanly alongside the muscle. After separation of the muscle capsule from Tenon's the muscle can be recessed and resected under Tenon's capsule without appreciably disturbing the check ligaments. In the closure, Tenon's capsule is approximated with one or two fine buried stitches. The conjunctiva is closed separately, and thereby its mobility is maintained. Postoperative reaction is minimal. Complications, such as scarring of the conjunctiva, granuloma formation, or retraction of the caruncle, have been less than when Tenon's capsule has been allowed to retract under the conjunctiva.

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ELECTRORETINOGRAPHY IN CIRCULATORY DISTURBANCES OF THE RETINA

II. The Electroretinogram in Cases of Occlusion of the Central Retinal Artery or of One of Its Branches

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ROTTERDAM, NETHERLANDS

IN HIS monograph, Karpe [1] describes two cases of so-called embolism of the retinal artery. In one case, in which there was an almost total obstruction of the circulation for seven days, the electrical response was subnormal, whereas an embolus in a small arterial branch during the acute stage of the disease gave rise only to an increased a-wave and a reduced b-wave—by definition, a negative electroretinogram. Once the circulation had been restored, it was observed that the negative electroretinogram had changed to a normal one. It seemed reasonable to assume, therefore, that the future development in a case of so-called embolism of the retinal artery could be predicted from a follow-up of the electroretinogram.

In a previous paper,[2] I have described a similar relation between the development of the disease and the changes in the electrical response in cases of occlusion of the retinal vein. The present article reports on 31 cases of occlusion of the central artery or of one of the retinal arteries, while, at the same time, it discusses vasodilatory therapy from an electroretinographic standpoint.

METHODS AND MATERIAL

Technique of Recording.—The recording technique was essentially the same as that described in the previous paper.[2]

Material.—The case histories of 24 patients with an occlusion of the central artery are given. One of the patients presented an occlusion in both eyes. Also included are the data on seven patients who had an occlusion of one or more branches of the central artery.

The Table summarizes the data on both groups as obtained during the first and last examination of each patient. The visual acuity, electroretinographic data, and description of the fundus picture are given for both the affected and the "normal" eye. The cases are grouped according to the duration of the complaints.

The time lapse between the two examinations varied considerably, owing to the fact that the final examinations were made shortly before this study terminated. In some cases the follow-up examination could not be performed at the moment planned because of the death or departure of the patient, so that data obtained at earlier examinations had to be included in the Table.

The distribution of the patients over the various age groups resembles that for the occlusion of the central retinal vein in my investigations.[2] The same similarity holds for the distribution of the sexes, as 17 men and only 7 women were affected (Fig. 1). As in my study on occlusion of the central vein, the arterial occlusion in most cases occurred at the age of 70 or over, suggesting a relationship to general senile arteriosclerosis.

From the Rotterdam Eye Hospital.

Clinical Data in Twenty-Four Cases of Occlusion of Central Retinal Artery and Seven Cases of Occlusion of One or More of Its Branches

						First Examination							Last Examination					
						Affected Eye			Normal Eye					Affected Eye			Normal Eye		
							Erg			Erg					Erg			Erg	
Case No.	Sex	Age, Yr.	Type of Occlusion *	Onset of Symptoms, Days		Visual Acuity	Type	b-Potential, Mv.	Visual Acuity	Type	b-Potential, Mv.	Fundus Picture	Time Lapse Since 1st Examination, Mo.	Visual Acuity	Type	b-Potential, Mv.	Visual Acuity	Type	b-Potential, Mv.
										Group 1									
1	M	75	.....	½	O.S.	1/60	Neg.—	0.11	10/10	Supern.	0.43	Normal	13	8/10	Neg.—	0.26	9/10	Norm.	0.36
2	M	44	.....	½	O.S.	1/∞	Neg.—	0.16	11/10	Neg.+	0.36	Normal	4	1/∞	Neg.—	0.13	11/10	Neg.+	0.42
3	M	72	.....	1	O.D.	1/300	Neg.—	0.15	9/10	Neg.—	0.11	Arteriosclerosis	..	.....	.....	....	.....	.....	....
4	M	44	.....	1	O.D.	1/∞	Neg.—	0.13	10/10	Neg.+	0.30	Normal	2	1/∞	Neg.—	0.12	10/10	Norm.	0.27
5	M	54	.....	1	O.D.	1/∞	Neg.—	0.19	10/10	Subn.	0.26	Normal	17	1/10	Subn.	0.17	10/10	Norm.	0.32
6	M	32	.....	1	O.S.	1/60	Neg.—	0.07	10/10	Norm.	0.31	Normal	26	5/10	Neg.—	0.14	10/10	Norm.	0.33
7	M	70	.....	1	O.D.	1/300	Neg.—	0.03	8/10	Neg.—	0.18	Arteriosclerotic hypertension	3	1/60	Neg.—	0.10	8/10	Neg.—	0.25
8	F	49	.....	1	O.S.	1/∞	Neg.—	0.17	8/10	Supern.	0.50	Hypertension	..	.....	.....	....	.....	.....	....
9	F	71	.....	1½	O.D.	1/∞	Neg.—	0.21	6/10	Neg.+	0.54	Hypertension	13	1/∞	Neg.—	0.14	6/10	Neg.+	0.45
10	M	74	.....	2	O.D.	1/∞	Subn.	0.14	9/10	Norm.	0.38	Hypertension	9	1/60	Neg.—	0.15	9/10	Subn.	0.27
11	M	63	.....	2	O.S.	1/∞	Neg.—	0.15	9/10	Neg.—	0.11	Normal	5	1/∞	Neg.—	0.28	10/10	Neg.—	0.29
12	M	74	.....	2	O.D.	1/∞	Neg.—	0.17	10/10	Supern.	0.52	Normal	..	.....	.....	....	.....	.....	....
13	F	29	.....	4	O.D.	1/300	Subn.	0.13	8/10	Neg.+	0.42	Normal	7	7/10	Subn.	0.14	8/10	Supern.	0.41
14	M	58	.....	4	O.D.	1/∞	Neg.—	0.24	10/10	Norm.	0.38	Normal	1	1/60	Subn.	0.14	10/10	Norm.	0.31
15	F	35	.....	4 (?)	O.S.	6/10	Neg.+	0.52	10/10	Supern.	0.48	Normal	..	.....	.....	....	.....	.....	....
16	M	73	.....	Some	O.D.	0	Neg.—	0.07	7/10	Norm.	0.38	Normal	5	0	Neg.—	0.06	7/10	Norm.	0.40
17	M	31	.....	9	O.D.	1/300	Subn.	0.25	1/60	Subn.	0.25	Normal (amblyopia)	29	10/10	Subn.	0.28	1/60	Subn.	0.21
18	M	61	.....	10	O.S.	1/60	Subn.	0.22	7/10	Subn.	0.17	Sclerosis of retinal and choroidal vessels	22	1/60	Subn.	0.19	5/10	Subn.	0.21
19	F	72	.....	10	O.D.	1/∞	Subn.	0.16	....	.....	....	.....	1	1/∞	Neg.+	0.31	.....	.....	....
					O.S.	4/10	Subn.	0.24	....	.....	....	.....	1	1/60	Norm.	0.38	.....	.....	....
20	M	81	.....	Several	O.S.	1/∞	Subn.	0.09	7/10	Subn.	0.12	Myopia gravior	5	1/∞	Subn.	0.14	6/10	Subn.	0.19
21	M	50	.....	Several	O.D.	9/10	Neg.—	0.27	9/10	Neg.+	0.31	Normal	13	9/10	Neg.+	0.32	9/10	Neg.+	0.30
22	M	61	.....	Several	O.D.	1/60	Neg.+	0.42	9/10	Neg.+	0.39	Slight arteriosclerosis	2	1/60	Neg.+	0.32	10/10	Neg.+	0.32
23	F	68	.....	2 wk.	O.D.	5/10	Neg.+	0.65	10/10	Supern.	0.58	Normal	6	5/10	Supern.	0.48	10/10	Supern.	0.58
24	F	77	.....	2 wk.	O.S.	0	Exting.	0	6/10	Norm.	0.36	Normal	8	0	Exting.	0	6/10	Norm.	0.34
										Group 2									
1	F	47	A. nas. inf.	1	O.S.	9/10	Neg.+	0.42	9/10	Neg.+	0.47	Normal	3	10/10	Neg.+	0.41	10/10	Neg.+	0.36
2	M	27	A. temp. inf.	2	O.D.	10/10	Norm.	0.39	10/10	Norm.	0.37	Normal	20	10/10	Norm.	0.41	10/10	Supern.	0.49
3	F	55	A. nas. and temp. inf.	3	O.S.	10/10	Neg.—	0.25	10/10	Neg.—	0.28	Slight hypertension-arteriosclerosis	15	10/10	Neg.—	0.27	10/10	Neg.+	0.30
4	M	28	A. temp. inf.	6	O.D.	9/10	Subn.	0.19	10/10	Subn.	0.09	Normal	31	10/10	Subn.	0.27	10/10	Norm.	0.36
5	F	17	A. temp. inf. and sup.	1 wk.	O.D.	10/10	Neg.+	0.37	10/10	Norm.	0.34	Normal	20	10/10	Norm.	0.35	10/10	Norm.	0.34
6	F	58	A. temp. sup.	2 wk.	O.D.	3/10	Neg.+	0.30	10/10	Neg.—	0.29	Normal	15	10/10	Norm.	0.31	10/10	Norm.	0.32
7	M	50	A. nas. sup.	2 wk.	O.D.	10/10	Norm.	0.35	10/10	Norm.	0.39	Slight hypertension	..	.....	.....	....	.....	.....	....

* Occlusion in Group 1 was of the central artery. The branch or branches involved are indicated in Group 2.

The electroretinograms of 23 patients could be followed regularly. However, in only three cases was the recording of the electroretinogram possible for more than one year; four patients were examined regularly over a period of more than half a year. The electroretinograms of 16 patients were recorded regularly over a period of less than half a year, whereas eight patients could be examined only sporadically.

There is, I believe, a significant difference between the patients with an occlusion of the central retinal vein and the patients reported on in this study. In the case of an arterial occlusion the patient realizes earlier the seriousness of his condition. After a relatively short period of treatment, he is apt to believe that his condition cannot be improved by any therapeutic measure. As the first symptoms (sudden loss or deterioration of visual acuity) are more acute than those of occlusion of the central retinal vein, it is clear that most patients come to the physician at an earlier stage of the disease.

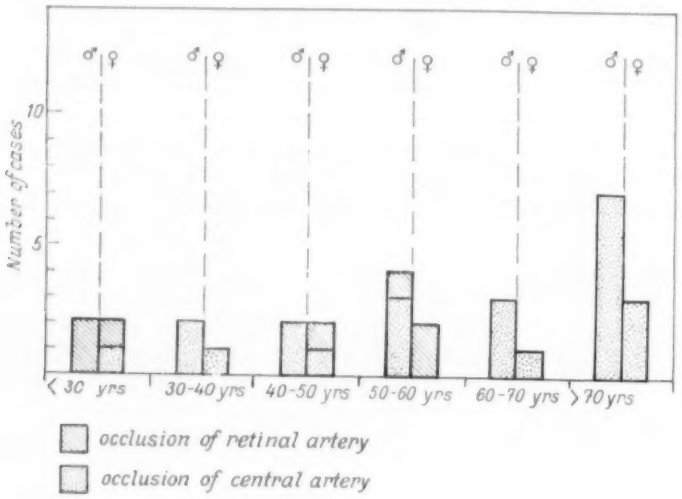

Fig. 1.—Cases representing the two types of arterial occlusion grouped according to age.

RESULTS

For the greater part of the eyes with an occlusion of the central retinal artery (Table, Group 1), I recorded a subnormal b-potential, usually combined with a pronounced a-wave. Most frequent in this group was the occurrence of the negative — type of electroretinogram, 21 patients showing a subnormal b-potential, as compared with 3 patients with a supernormal b-potential. These three patients, at the same time, showed a prominent a-wave—by definition, a negative + electroretinogram. Two of these three patients had rather good central vision, whereas of the 21 patients showing a negative — or a subnormal electroretinogram, only 1 had good central vision.

It is of interest that in spite of the very poor visual function in this group of 21 cases, in only 1 could no electroretinogram be recorded ("extinguished" type, Karpe). In that case, however, trauma to the ophthalmic artery caused not only ischemia of the retina, but also an obstruction of the ciliary circulation. This case supports the opinion that even in a total obstruction of the central retinal artery

accompanied with total loss of vision, the normally intact ciliary circulation is capable of supplying the anterior parts of the retina with blood to such a degree that a measurable b-potential can be recorded. This is in accordance with the clinical phenomenon of the limitation of the edema and the necrosis to the posterior pole of the eye in cases of occlusion of the central artery.[8]

In the eyes showing an occlusion of one of the branches of the central artery, the normal or supernormal b-potentials are dominating. Group 2 (Table) thus shows a normal or supernormal b-potential for five patients and a subnormal b-poten-

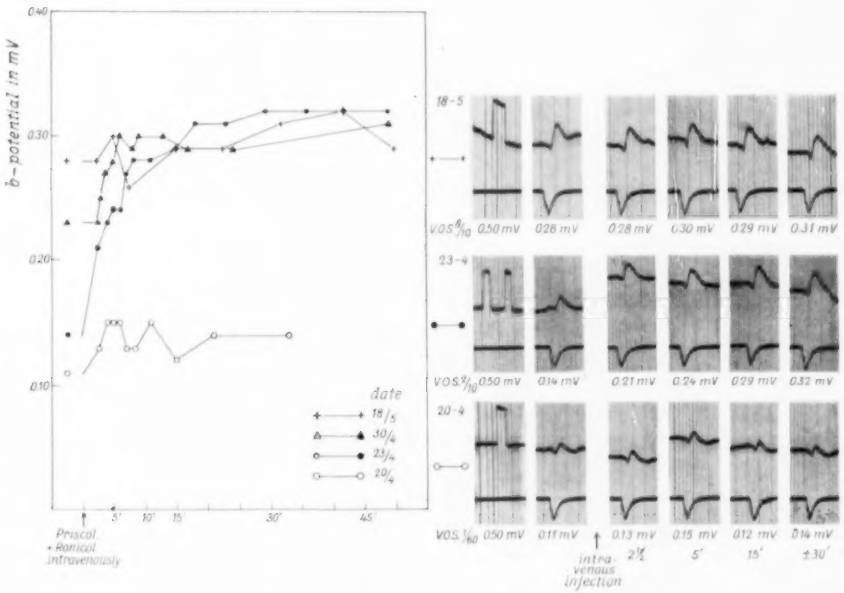

Fig. 2.—Influence of vasodilator therapy on the amplitude of the b-potential recorded in the affected eye of a patient with an occlusion of the central retinal artery. No significant rise in b-potential could be registered in the subnormal b-potential after the intravenous injection of a mixture of tolazoline hydrochloride 0.020 gm., and Roniacol, 0.100 gm. (line of circles; 20/4). After a few days of vasodilatory therapy, a marked improvement in the electrical response, as well as in the visual acuity, could be observed. No alteration was found in the pronounced a-wave. Samples of the electroretinograms recorded are reproduced at the right of the curves; the lower tracings represent the light flash recorded.

tial for two patients. In four patients a pronounced a-wave was observed. The data summarized in this group, however, are much too limited to permit of any definite conclusion regarding the supposition that the negative + type of electroretinogram is representative of this group.

Group 1.—It is interesting to note that in many patients with an occlusion of the central retinal artery the electroretinogram of the unaffected eye followed the same pattern as that of the affected eye. In only 5 of 23 patients (22%) did the unaffected eye show a normal electroretinogram, whereas in at least 14 patients (61%) a normal fundus picture was observed. Of five of seven patients with occlusion of one

of the branch arteries the ophthalmoscopic picture of whose unaffected eye was found to be normal, the electroretinogram was normal for only three.

These facts support the opinion that electroretinography can reveal a circulatory disturbance in a number of cases in which the ophthalmoscope does not show any abnormality.

For several patients who were examined regularly with the electroretinographic technique, curves were drawn illustrating the course of the electroretinogram and the visual acuity during and after treatment.

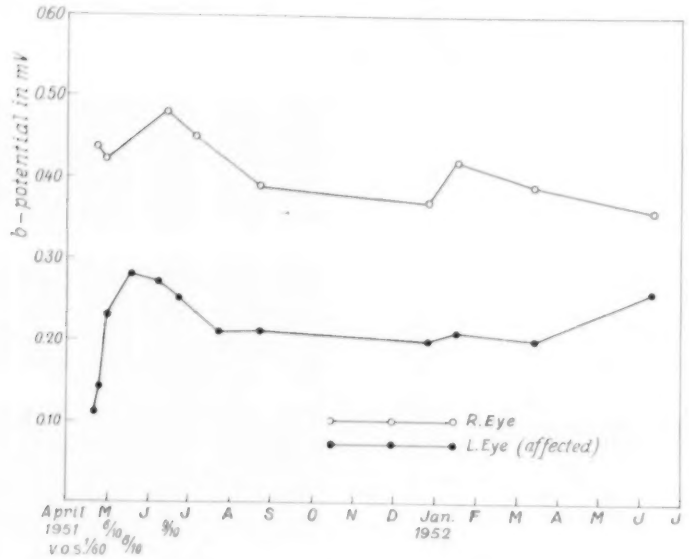

Fig. 3.—Curves showing the course of the b-potential in the patient represented in Figure 2. Intensive vasodilator therapy improved the b-potential considerably in the affected (left) eye. Improvement in the visual acuity coincided with an increase in the b-potential. Reduction in b-potential is shown following suppression of intensive administration of vasodilators. Normalization of the a-wave was not obtained.

A discussion of the results from an electroretinographic standpoint follows:

Case 1.—B. H. complained of blurred vision of the left eye, which had started suddenly a few hours before the first examination. Ophthalmoscopic examination revealed the well-known picture of occlusion of the central artery.

Electroretinography revealed a marked subnormal b-potential in the affected eye, whereas the other eye showed a somewhat higher than normal b-potential. As soon as possible, an intravenous injection of tolazoline hydrochloride, 0.020 gm. and Roniacol tartrate, (β-pyridil carbinol tartrate; Ronicol), 0.100 gm., was administered. After the injection the electroretinogram of the left eye was recorded regularly over a period of half an hour. In Figure 2 the course of the b-potential is plotted against time, in minutes, after the injection. As can be seen, no significant improvement in the b-potential followed the first injection (line of circles, Fig. 2). The injection therapy was continued during the next six hours, with three injections of the aforementioned mixture of tolazoline and Roniacol. During the following days there was some improvement in the visual acuity. As I followed daily the course of the b-potential after the injections, I observed

that after a few days the amplitude of the b-potential started to rise considerably after the injection (line of dots, Fig. 2). The base values for the b-potential which were registered each morning before the injection therapy was begun were rising, too, as a result of which the sharp initial increase in the b-potential following the injection gradually decreased. The curves show that, in spite of the strong vasodilation, the b-potential could not be brought up to a higher level than approximately 0.30 mv. The visual acuity improved in a few weeks to rather normal values.

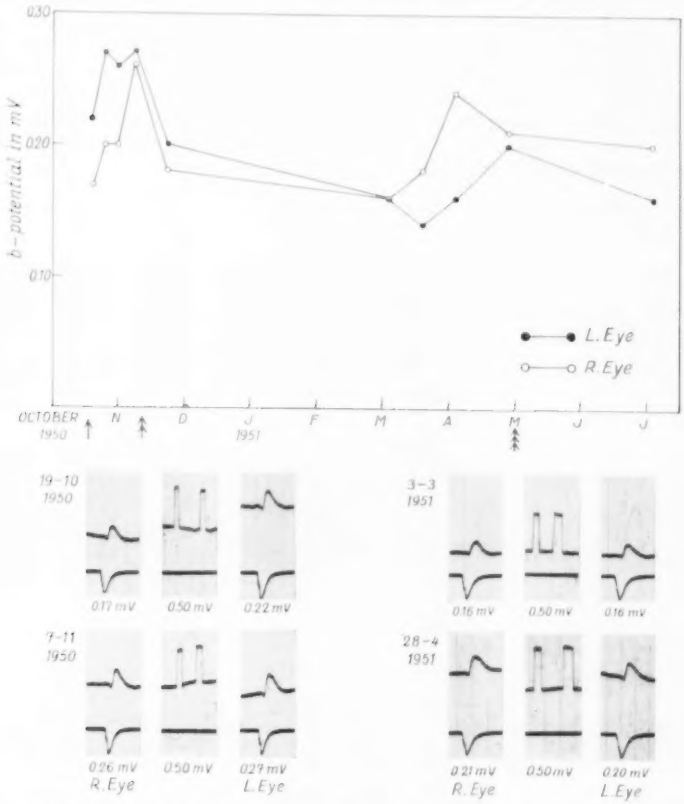

Fig. 4.—Curves showing the b-potentials in both eyes, plotted against time, in months, in a patient with an occlusion of the central retinal artery of the left eye due to general arteriosclerosis. The right eye showed extreme sclerosis of both the retinal and the choroidal vessels. Discharge from the hospital was followed by reduction of the b-potential (double arrow), which had shown improvement during intensive vasodilator therapy. All treatment was stopped (triple arrow) after further vasodilator therapy, given orally for almost half a year. The b-potentials remained on the same level. The a-wave was less pronounced than at the start of the therapy.

It is believed that this striking success, which, however, was obtained in only a few cases, could have been predicted from the improvement in the b-potential following a few days of vasodilator therapy.

As it was found in this case that the b-potential kept on rising after the five-minute period of dark adaptation, which is usually considered sufficient for it to reach a stationary condition, the electroretinographic recording in this and in the other cases mentioned in the present investigation was made only after at least 30 minutes of complete rest in the dark.

Figure 2 shows, in addition to the curves discussed, some samples of the electroretinograms recorded on the dates noted.

In figure 3 are drawn the b-potential curves for the same patient in relation to time, expressed in months. The marked rise in the base values of the b-potential of the left retina during the first weeks of vasodilator therapy is worth mentioning. After one month the intravenous injections were replaced by oral administration of a dilator (tolazoline; Roniacol). In the beginning of 1952 all therapy was stopped. The electroretinograms did not show any significant deterioration in their b-potentials.

Continued electroretinographic observation will be necessary in order to judge whether there is an adequate retinal circulation in the future. The ophthalmoscopic picture of the sound eye did not change in the course of the disease, whereas the b-potential showed a trend toward normal.

Case 18.—The electroretinographic history of P. K. is also of interest. On consulting his ophthalmologist, this man complained of very blurred vision in his left eye for 10 days. The fundus revealed a picture of occlusion of the central artery. The unaffected eye showed extreme atrophy of the retina and the choroid. Only a very small island, including the macula, was left intact. The visual field was extremely narrowed.

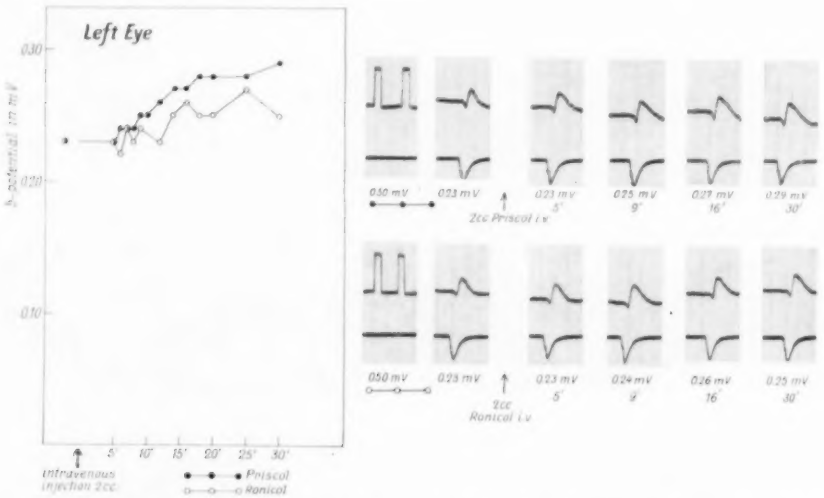

Fig. 5.—Influence of a single injection of tolazoline hydrochloride, 0.020 gm., and Roniacol tartrate, 0.100 gm., given intravenously, in the patient represented in Figure 4. Increase of the b-potential is not very marked, as a consequence of the extreme sclerosis of the vessels.

Therapy was started with intravenous injection of a mixture of tolazoline hydrochloride (0.020 gm.) and Roniacol tartrate (0.100 gm.) twice a day.

Figure 4 illustrates the course of the b-potentials of the electroretinogram of both eyes plotted against time, in months. During the hospitalization intensive vasodilator therapy was given. The b-potentials increased, and a fairly pronounced a-wave was recorded. After his discharge from the hospital (double arrow) the a- and b-waves again decreased in height. Vasodilator therapy was then resumed orally. Therapy was discontinued several months later (triple arrow). The b-potentials remained rather constant. At that moment the a-waves were scarcely visible.

Owing to the extreme sclerosis of the choroidal vessels, it seemed unlikely that normal values for the b-potential could be regained by any therapeutic measure whatever. In cases in which the sclerotic process is not extremely developed, it has been shown that a normal value for the b-potential can be regained with vasodilator therapy.[4]

In Figure 5 the effects on the electroretinographic records of the affected eye of the two vasodilators used in this investigation are shown separately. It is interesting to note that intravenous injection of tolazoline or Roniacol alone had only a slight effect on the course of the b-potential.

CASE 23.—A. E. had an occlusion of the central retinal artery of the right eye, coupled with general arteriosclerosis, Grade 3, and hypertension. The electroretinogram was of the negative + type. The first examination showed that the affected eye possessed fairly good central vision. Ophthalmoscopic study revealed marked arteriosclerosis of the retinal vessels, as shown by

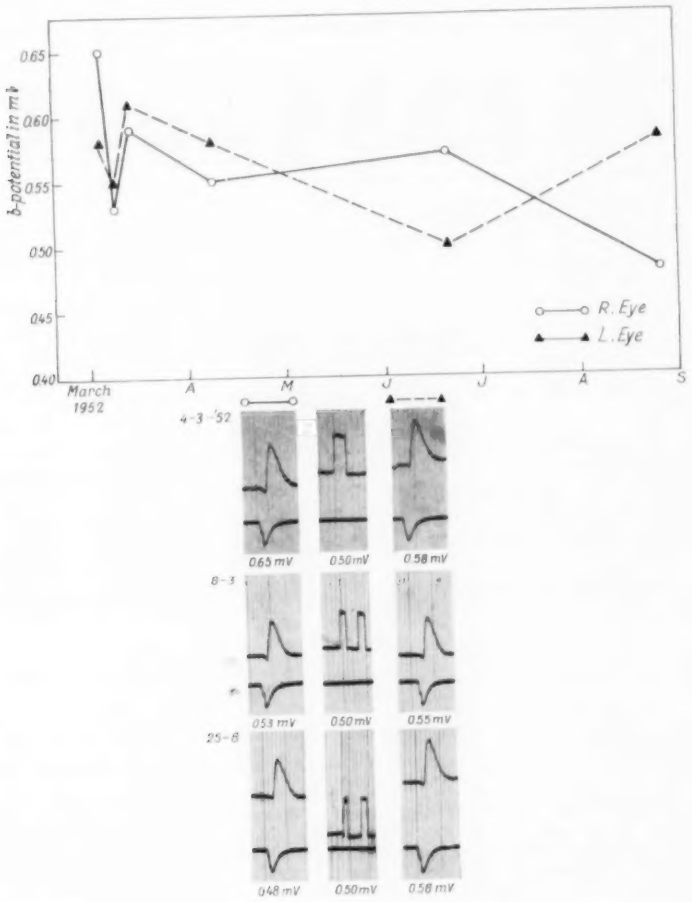

Fig. 6.—Curves showing the course of the b-potentials plotted against time, in months, for a patient with an occlusion of the central retinal artery in the right eye. In the course of the treatment a reduction took place in the supernormal a-potential, as well as in the b-wave, of the affected eye.

arteriovenous crossings, some hemorrhages, and variations in caliber of the vessels. Slight edema of the optic disk and of the retina at the posterior pole was observed. The unaffected eye presented no signs of an arteriosclerotic and hypertensive stage in the retina, although the electroretinogram showed an abnormally high b-potential, without a clear a-wave.

The extremely high value for the b-potential of the affected eye showed some reduction in the course of the treatment, which consisted of intravenous injections of Roniacol, 0.100 gm. twice

a day, combined with oral administration of the drug, 0.050 gm. three times a day. Figure 6 shows the course of the b-potentials of both eyes, together with samples of records taken on several dates. After his discharge from the hospital, a few weeks later, the administration of Roniacol was continued orally for three months.

Owing to the fixed sclerotic condition of the choroidal and retinal vessels, a full normalization of the electroretinogram in the future is not to be expected. The hypertension, however, has improved since the customary treatment was instituted (rest in bed, saltless diet, etc.). Coin-

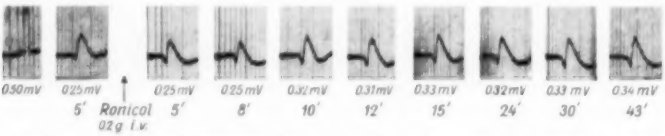

Fig. 7.—Follow-up electroretinogram of the left retina in a patient with an occlusion of two branches of the central artery. Intravenous injection of Roniacol, 0.100 gm., led to an increase in the b-potential, producing a change from a negative — to a negative + electroretinogram.

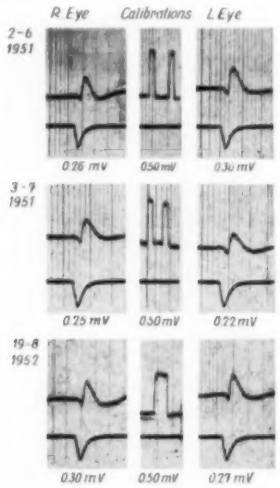

Fig. 8.—Electroretinograms of the same patient as the one referred to in Figure 7. The electroretinograms of the right eye are reproduced to the left of calibrations of 0.50 mv. to 1 cm. The a-waves on both eyes are well defined. No significant change appeared in the electroretinogram during treatment or afterward, during a period of over one year. The left eye showed minor signs of an arteriosclerotic-hypertension fundus.

ciding therewith was an improvement in the metabolic condition of the retina, demonstrated by a reduction in the amplitude of the a-wave. The change in the type of electroretinogram, from negative + to supernormal, followed this improvement.

I believe that the supernormal b-potential in this case is the reflection of a circulatory disturbance in the retinas. The retinal elements were not deprived of their circulation to such a degree, however, that as a consequence a subnormal b-potential was recorded. A marked reduction in the visual acuity can, then, be expected in the future.

The electroretinographic study of the apparently normal eye provides valuable information regarding the retinal circulation in general. Although no pathological

changes could be seen with the ophthalmoscope, I suspect the presence of minor circulatory disturbances in the retina, as the electroretinogram showed a supernormal b-potential. Neither the visual function nor the fundus picture, however, is, in my opinion, a decisive indicator of the ultimate condition of the retinal circulatory apparatus.

GROUP 2.—Cases of occlusion of one or more branches of the central retinal artery.

CASE 3.—M. S., who had complained of blurred vision of the left eye for three days, showed localized edema of the retina following occlusion of both the nasal and the temporal inferior arteries. In the left eye signs pointed to a minor hypertensive-arteriosclerotic condition of the retinal vessels. After an electroretinographic recording, Roniacol (0.100 gm.) was given intravenously. The electroretinogram of the affected eye was then recorded for 52 minutes after the injection. Abnormal electroretinograms for both eyes, demonstrated by the pronounced a-waves, pointed to a pathological circulation, differing in degree, in the two retinas. This patient had general arteriosclerosis, Grade 3, combined with hypertension.

Figure 7 illustrates the changes in the electroretinogram following vasodilation. The b-potential gained normal values, whereas the a-potential did not show much alteration.

A follow-up of the electroretinograms over a longer period was made. In the course of the treatment neither the a-potentials nor the b-potentials showed lasting improvement. When, a few months later, vasodilator treatment was stopped, the values for the a- and b-potentials did not show much change. An electroretinogram one year later did not reveal any further alterations (Fig. 8).

CASE 4.—N. I., with an occlusion of the temporal inferior artery, presented peculiar electroretinographic effects. The b-potentials of both eyes were found to be subnormal, whereas the normal eye did not show the least sign of any abnormality in visual function or in the fundus picture. This young patient, aged 28, had general arteriosclerosis. After intensive vasodilator therapy the b-potential returned to normal. The electroretinographic observations have been described in a previous paper.[5] It was possible to follow this patient over 31 months. The b-potential has remained normal ever since the therapy was stopped, two years ago.

COMMENT AND CONCLUSIONS

The data summarized in the Table and the case histories indicate that an apparently normal fundus picture (in the sound eye) may be associated with marked abnormalities in the electroretinogram. I am convinced that such an abnormal electrical response is connected in some way with an abnormal metabolic condition of the involved retina, which has thus far failed to produce a pathological ophthalmoscopic picture.

Even when the normal fundus picture has changed, major variations in the electroretinogram can be registered without apparent change in the visual function or in the ophthalmoscopic picture of the involved retina. In this connection, reference is made to an earlier article,[2] in which the case of a patient with retinal hypertension is described. In this case a disturbance in the metabolic state of the retina was indicated by the complete temporary disappearance of the a- and b-potentials of the electroretinogram, although, at the same time, the retinal function was fully preserved and the fundus picture was unaltered.

I accept the idea that a supernormal electroretinogram is the response of the retinal elements in a state of increased irritability following deterioration of the metabolic state of the retina. The supernormal response can be turned into a normal one by therapeutic measures, or it can be changed to a subnormal electroretinogram by the further deterioration of the metabolic condition. Even then, it is possible in

some cases to produce a restoration of the electrical activity, through vasodilation for instance, in cases of nonfixed sclerosis of the vessels involved.

The effects on visual acuity of treatment of occlusion of the central artery have so far not been very encouraging; only in 4 of 21 cases in which the visual acuity was 1/60 or less and the electroretinogram negative + or subnormal could improvement in the visual acuity of up to 5/10 or more be recorded after intensive vasodilator therapy. Some improvement (to 1/60) could be shown in 4 cases, whereas in 10 cases no improvement at all in the visual acuity appeared.

In fresh cases (of up to four days after the beginning of symptoms) the results of treatment were better than in the older ones; in 7 of 11 of the fresh cases visual acuity improved, whereas of the seven older ones, improvement occurred in only one.

The electroretinogram showed improvement in four cases in the first group, of the fresh cases, as against one in the second group. However, a close correlation between improvement in visual acuity and electroretinographic data is not to be expected, as these two functions cannot be compared: the first is a function of the macular area and the second a function of the retinal circulation as a whole.

Application of early vasodilator therapy is of major importance. The most potent vasodilator therapy known at present consists of a combination of tolazoline hydrochloride (0.020 gm.) and Roniacol tartrate (0.100 gm.) given intravenously. I believe that evaluation of such a therapy must be made on the basis not only of changes in visual acuity, visual fields, and fundus picture but, preferably, of the data obtained by electroretinography.

The vasodilator therapy, however, is only part of the medical therapy, which must be directed, in the first place, toward improvement in the general condition of the patient. General and dietetic measures and, eventually, treatment of the insufficient general circulation, must be instituted, if necessary.

SUMMARY

The electroretinograms in 24 cases of occlusion of the central retinal artery and in 7 cases of occlusion of a branch artery, taken before, during, and after treatment, are discussed. In several cases follow-up examinations could be performed for periods of several months to more than two years.

It is concluded that a negative — electroretinogram must be considered a typical symptom in cases of occlusion of the central artery. There is some evidence that the negative + type is the one which predominates in cases of occlusion of a branch artery.

In a large number of cases electroretinography revealed abnormalities in the apparently normal eye which were not shown by alteration in function or in the fundus picture.

Curves showing the course of the a- and b-potentials over longer periods are presented to give a better appreciation of the state of the retinal circulation.

Treatment must be directed, in the first place, toward improvement in the general condition; this likewise benefits the sound eye.

Vasodilator therapy must be applied as soon as possible, as the data indicate that success of the treatment depends largely on the time lapse between the first signs of the disease and the beginning of the intensive vasodilator treatments.

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ELECTRORETINOGRAPHY IN CIRCULATORY DISTURBANCES OF THE RETINA

III. Electroretinogram in Cases of Senile Degeneration of the Macular Area

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IT IS GENERALLY accepted that senile degeneration of the macula results from pathological alterations in the choroid and/or retinal vessels. As such alterations affect the electroretinal response of the eye, it was considered of interest to study my electroretinographic data pertaining to patients with this condition. The present article reports the results of a study of 80 cases of senile degeneration of the macular area. The electroretinographic technique was the same as that described in detail in an earlier paper.[1]

MATERIAL

In the accompanying Table is given a summary of the data on sex, age, visual acuity, and electroretinographic findings in both eyes in 80 cases of senile degeneration of the macula. The data are presented in seven groups: (1) senile disciform degeneration, 16 cases; (2) senile chorioretinal degeneration (Haab), 17 cases; (3) senile cystoid degeneration, 5 cases; (4) senile colloid degeneration, 4 cases; (5) senile circinate degeneration, 6 cases; (6) senile degeneration accompanied by minor signs of arteriosclerosis and/or hypertension of the retinal vessels, 13 cases, and (7) unclassified cases of senile degeneration, 19 cases.

It is well known that it is often difficult to classify cases of macular disease satisfactorily. In some the type of macular involvement differs in the two eyes. In others the type of macular disturbance changes during the course of the disease. This explains why a rather large number of cases is found in the unclassified group.

RESULTS

In the following pages I shall discuss in some detail cases belonging to the various groups, with particular reference to the discrepancy between the ophthalmoscopic picture and the electroretinogram. A discussion of the results of treatment is included.

GROUP 1.—*Senile Disciform Degeneration of the Macula.*

CASE 13.—F. C. had complained of decreasing vision in his right eye for several months. For some weeks the vision in his left eye had also been deteriorating. The condition was diagnosed as disciform degeneration of the macular region in both eyes, the left eye being less affected than the right. General examination revealed pronounced arteriosclerosis. No further pathological condition was found. Other than the macular affection, resulting from localized sclerosis of the choriocapillaris, no pathological signs were visible ophthalmoscopically; the retinal vessels showed no changes resulting from arteriosclerosis and/or hypertension.

Therapy was started with papaverine, 60 mg. taken orally three times a day. Various other vasodilators were tried, but further deterioration of the visual acuity in the left eye could not be prevented. The electroretinogram showed a subnormal b-potential, which did not change much in the course of the disease (Fig. 1).

Clinical Data in Eighty Cases of Senile Degeneration of the Macular Area

Case No.	Sex	Age, Yr.	(More) Affected Eye: Visual Acuity	(More) Affected Eye: ERG Type*	(More) Affected Eye: ERG b-Potential, Mv.	Normal, or Less Affected Eye: Visual Acuity	Normal, or Less Affected Eye: ERG Type*	Normal, or Less Affected Eye: ERG b-Potential, Mv.
Senile Disciform Degeneration								
1	M	59	1/60	Neg.	0.25	8/10	Neg.+	0.41
2	M	66	2/60	Subn.	0.23	2/10	Neg.—	0.20
3	M	68	1/60	Subn.	0.10	9/10	Norm.	0.26
4	F	70	1/10	Subn.	0.17	5/10	Norm.	0.29
5	F	71	1/60	Subn.	0.21	4/10	Norm.	0.26
6	F	72	1/60	Subn.	0.22	8/10	Norm.	0.26
7	M	73	3/60	Subn.	0.19	3/60	Norm.	0.28
8	M	77	1/60	Subn.	0.19	9/10	Subn.	0.17
9	M	77	1/300	Norm.	0.30	9/10	Norm.	0.35
10	M	78	1/60	Neg.—	0.29	....	.....	....
11	F	79	3/60	Subn.	0.23	....	.....	....
12	M	79	2/60	Subn.	0.10	2/60	Subn.	0.14
13	M	81	1/60	Subn.	0.20	5/10	Subn.	0.20
14	F	81	2/300	Norm.	0.37	5/10	Norm.	0.41
15	M	83	1/60	Subn.	0.19	9/10	Norm.	0.30
16	M	88	1/60	Neg.—	0.16	6/10	Norm.	0.25
Senile Chorioretinal Degeneration								
1	M	54	2/10	Subn.	0.12	3/10	Subn.	0.10
2	F	55	2/60	Norm.	0.29	4/10	Norm.	0.34
3	F	70	1/60	Norm.	0.36	3/60	Norm.	0.32
4	F	70	6/10	Norm.	0.29	6/10	Norm.	0.39
5	M	73	1/60	Subn.	0.15	9/10	Subn.	0.23
6	M	73	4/10	Norm.	0.37	6/10	Norm.	0.37
7	F	73	4/10	Subn.	0.12	9/10	Subn.	0.12
8	M	75	6/10	Subn.	0.24	9/10	Norm.	0.28
9	M	76	1/60	Exting.	0	2/60	Exting.	0
10	M	76	7/10	Norm.	0.25	10/10	Subn.	0.21
11	M	76	2/10	Norm.	0.29	4/10	Norm.	0.27
12	M	79	2/60	Subn.	0.10	5/10	Subn.	0.13
13	F	80	1/60	Subn.	0.22	2/60	Subn.	0.21
14	M	83	1/60	Norm.	0.25	3/10	Subn.	0.14
15	M	83	5/10	Subn.	0.08	5/10	Subn.	0.07
16	M	83	7/10	Subn.	0.14	8/10	Subn.	0.15
17	M	87	1/60	Subn.	0.23	1/10	Exting.	0
Senile Cystoid Degeneration								
1	F	68	4/10	Neg.+	0.47	6/10	Norm.	0.35
2	F	69	1/10	Norm.	0.37	6/10	Subn.	0.18
3	F	70	3/60	Norm.	0.37	3/60	Norm.	0.40
4	M	73	1/10	Subn.	0.22	9/10	Subn.	0.20
5	F	79	1/60	Supern.	0.48	4/10	Norm.	0.34
Senile Colloid Degeneration								
1	M	67	2/60	Subn.	0.19	8/10	Subn.	0.24
2	F	73	7/10	Norm.	0.38	8/10	Norm.	0.37
3	M	76	7/10	Subn.	0.23	7/10	Subn.	0.23
4	M	84	1/60	Norm.	0.30	5/10	Norm.	0.27
Senile Circinate Degeneration								
1	F	53	4/300	Supern.	0.42	10/10	Norm.	0.35
2	M	67	4/60	Subn.	0.19	10/10	Norm.	0.25
3	F	71	4/10	Norm.	0.29	8/10	Norm.	0.34
4	F	74	4/10	Norm.	0.34	5/10	Norm.	0.34
5	F	76	8/10	Supern.	0.51	8/10	Supern.	0.55
6	M	78	2/60	Neg.+	0.38	2/60	Neg.+	0.39
Senile Degeneration with Arteriosclerosis of Retinal Vessels								
1	M	60	1/60	Neg.+	0.51	10/10	Neg.+	0.35
2	F	60	6/10	Norm.	0.36	9/10	Norm.	0.35
3	F	60	3/10	Supern.	0.44	5/10	Supern.	0.45
4	F	64	6/10	Neg.+	0.51	6/10	Neg.+	0.51
5	F	65	2/60	Neg.+	0.42	9/10	Norm.	0.33
6	F	68	6/10	Supern.	0.44	7/10	Norm.	0.35
7	M	70	6/10	Supern.	0.43	7/10	Norm.	0.38
8	M	72	2/10	Neg.+	0.44	5/10	Neg.+	0.37
9	F	73	5/10	Neg.+	0.37	5/10	Neg.+	0.39
10	F	73	1/60	Norm.	0.32	10/10	Norm.	0.34
11	F	74	1/300	Neg.—	0.11	1/60	Norm.	0.38
12	M	76	7/10	Neg.+	0.32	10/10	Neg.+	0.34
13	F	79	1/10	Supern.	0.50	9/10	Norm.	0.40
Unclassified								
1	F	50	2/10	Norm.	0.33	6/10	Norm.	0.34
2	M	53	1/10	Norm.	0.37	10/10	Norm.	0.37
3	F	54	3/10	Norm.	0.26	5/10	Norm.	0.35
4	M	56	3/10	Supern.	0.53	10/10	Supern.	0.43
5	M	56	4/10	Norm.	0.29	10/10	Norm.	0.32
6	F	58	2/10	Norm.	0.39	7/10	Supern.	0.49
7	M	59	1/60	Subn.	0.19	5/10	Subn.	0.22
8	M	61	2/10	Supern.	0.43	10/10	Supern.	0.43
9	M	63	1/60	Norm.	0.32	5/10	Norm.	0.37
10	F	65	4/10	Supern.	0.52	9/10	Supern.	0.57
11	F	66	1/10	Norm.	0.28	6/10	Norm.	0.28
12	M	66	9/10	Supern.	0.42	10/10	Supern.	0.44
13	M	66	4/10	Norm.	0.30	6/10	Norm.	0.32
14	F	66	1/60	Norm.	0.37	4/10	Norm.	0.30
15	M	67	5/10	Norm.	0.30	5/10	Norm.	0.38
16	M	77	1/300	Norm.	0.25	2/10	Norm.	0.29
17	F	77	2/10	Subn.	0.23	3/10	Norm.	0.32
18	M	79	1/60	Subn.	0.19	5/10	Subn.	0.22
19	M	81	1/60	Norm.	0.32	5/10	Norm.	0.35

* Types of the electroretinogram are normal, subnormal, supernormal, negative+, negative, negative—, and extinguished.

I found that an extension of the standard period of five minutes of dark adaptation before recording of the electroretinogram is of importance when the effect of vasodilatory therapy is studied. A further increase in the b-potentials is sometimes obtained when this period of rest in the dark is increased up to 30 minutes. This increase is particularly noticeable in cases of disturbances in the retinal and/or choroidal circulation.[2]

Figure 2 shows improvement in the b-potential of the left eye of this patient after a prolonged period of dark adaptation. Figure 3 illustrates the effect of intravenous vasodilation

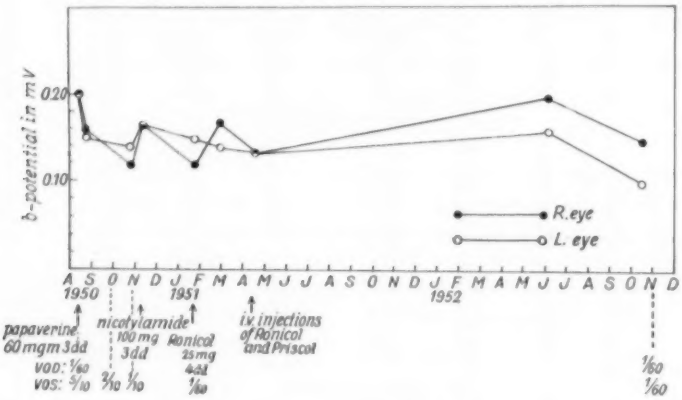

Fig. 1.—Curves showing the course of the b-potential in both eyes of a patient with senile degeneration of the disciform type, the right eye showing the more advanced stage of the macular affection. Visual acuity was reduced in spite of vasodilator therapy. No improvement could be recorded in the values for the b-potentials of the electroretinograms.

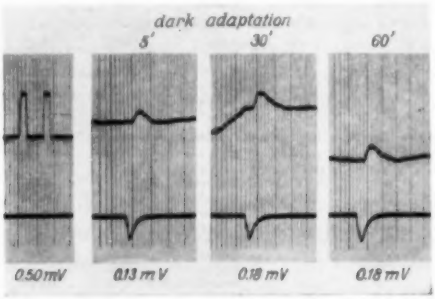

Fig. 2.—Influence of prolonged rest in the dark, viz., minor increase in the b-potential of the left eye, in the same patient as the one represented in Figure 1.

therapy on the b-potential of the left eye after a period of 45 minutes of dark adaptation. Tolazoline (Priscol), as well as β-pyridrol carbinol (Roniacol; Ronicol), produced some improvement, which, however, I do not consider significant.

When the b-potential does not show an increase following injection of a vasodilator substance, this may mean (*a*) that b-potential recorded lies within the normal limits of the age group under discussion, or (*b*) that the b-potential cannot show a significant increase because the retinal elements, apart from those in the macular area, are irreversibly changed, though neither the ophthalmoscope nor the study of

the visual fields can show any change in the periphery of the retina. In cases of arteriosclerosis and/or hypertension reversible changes in the b-potential have been observed rather frequently, even when the fundus picture did not show any signs of such a change (Henkes [2]). The retinal elements—photoreceptors, depending

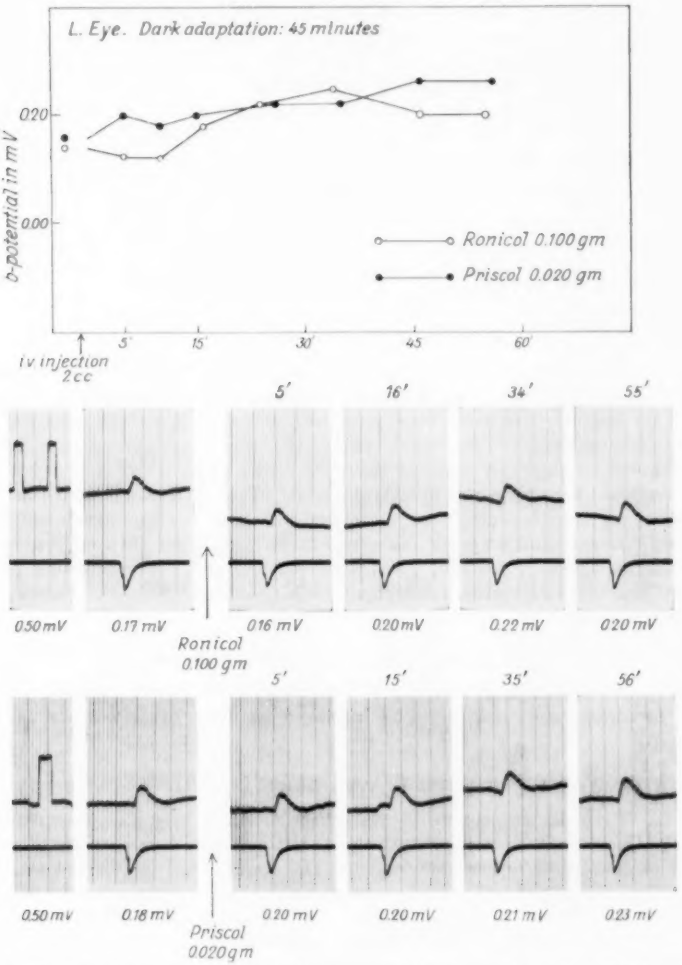

Fig. 3.—Curves showing the effect of intravenous injection of tolazoline, 0.020 gm., and of Roniacol, 0.100 gm., on the b-potentials of the left eye after dark adaptation of 45 minutes, in the same patient as the one represented in Figure 1.

on the choroidal circulation, or neurones of the second order, provided by the retinal circulation—are capable of showing an improvement in electrical response after intensive vasodilation, even to such a degree that markedly subnormal b-potentials can be converted into normal ones—sometimes, however, only after a prolonged period of vasodilation.[2c] In the case of the patient under discussion,

who did not show a significant increase in the b-potential, it must be assumed that a fixed arteriosclerotic choroidal circulation was present, responsible for the subnormal values of the electroretinograms recorded in both eyes.

Case 10.—J. L., with anophthalmos of the right eye, showed a typical disciform degeneration of the left macula. There were slight signs of arteriosclerosis of the retinal vessels, namely, tortuosity, enlargement of the vessels, and some irregularlity of the lumen. The electroretinograms, recorded after the usual period of five minutes of dark adaptation, varied considerably over a period of approximately two years. The visual acuity, visual fields, and ophthalmoscopic picture were constant. The electroretinographic findings were subnormal or normal, as tabulated, changing into a supernormal type, with a b-potential of 0.45 mv. after 30 minutes of dark adaptation and rest. Such a variability in the b-potential, not found in normal subjects, points in my opinion to reversible changes in the electrical response of the retinal elements following variations in the retinal circulation. With the ophthalmoscope these changes could not be observed.

Date	Type of Electroretinogram	b-Potential, Mv.
Jan., 1951	Normal	0.29
March, 1951	Subnormal	0.18
April, 1951	Normal	0.31
Dec., 1952	Normal	0.32

Case 1.—The data on C. B. are of particular interest. The right eye had been affected for a year and a half; the left eye, for half a year. Both eyes showed degeneration of the macular region, of the disciform type, with large cystoid and circinate degenerative fields, hemorrhages, and slight sclerotic changes in the retinal vessels. The left eye was affected to a much smaller degree than was the right eye. Figure 4 shows the course of the b-potentials in both eyes. In the long run there were some variations in the height of the b-potential, but on the whole the electroretinogram did not change to any considerable extent. The variations could not be put down to differences in lengths of the rest period in the dark before the electroretinogram was recorded. Vasodilation (tolazoline, given intravenously) did not change the b-potentials, either. In the course of the disease neither the visual acuity nor the fundus picture underwent further deterioration.

Factors in other types of senile degeneration must, therefore, be held responsible for some of the higher-than-normal b-potentials in cases of senile disciform degeneration of arteriosclerotic origin. The influence of these other factors will be discussed later.

Group 2.—*Senile Chorioretinal Degeneration (Haab).*

Case 12.—The electroretinograms of this patient (M. M.) have been discussed in part in a previous paper,[2c] but the development of the disease and the follow-up study of the electroretinographic data justify further consideration. This patient could be followed for over three years. Implantation of placenta and injections of cod liver oil had been used in an attempt to arrest the development of the macular process. The subnormal electroretinogram in both eyes did not improve, however. A considerable rise in the b-potential was recorded after vasodilation. Visual acuity in the less affected (left) eye was deteriorating in spite of continuous vasodilation. Over a period of two years the patient was given papaverine, nicotinoamide, Manganocholine (bis-[acetylcholine]-chloromanganate), tolazoline, and Roniacol. The electroretinogram did not show much deterioration over this period. There was even improvement in the height of the b-potentials following a prolonged stay in the dark (Fig. 5), and further improvement occurred when, after the 30 minutes of dark adaptation, an intravenous vasodilator was administered. Figure 6 illustrates the influence of tolazoline hydrochloride, 0.020 gm., and of Roniacol tartrate, 0.100 gm., on the b-potential of the electroretinogram of the left eye. In this case Roniacol proved to be somewhat superior to tolazoline, while both drugs were better than the other vasodilators administered orally (papaverine, 180 mg.; nicotinamide, 300 mg.;

Manganocholine, 300 mg.; Cyclospasmol [the 3,5,5-trimethylcyclohexamol ester of mandelic acid], 60 mg. a day). It is interesting to note that the b-potential in the course of the vasodilator period (June, 1949, until April, 1951) could be raised from 0.10 to 0.27 mv. in the right eye and from 0.13 to 0.40 mv. in the left eye. Subsequently, the b-potentials were of approximately the same height, even when all treatment was stopped. Though an optimal supply of the retina of the right eye was not obtained, owing to the arteriosclerosis of the choroidal circulation, the

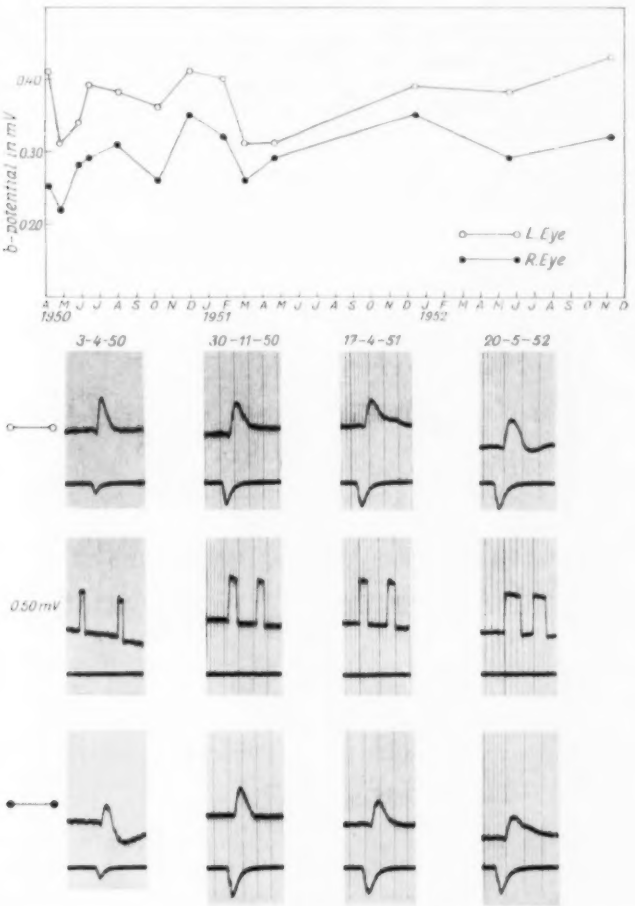

Fig. 4.—Curves illustrating the course of the b-potentials, recorded over a longer period, in a case of senile degeneration of a combined type. Specimens of electroretinograms are shown below.

relative high b-potentials after discontinuation of treatment justify the optimistic prognosis of restoration of the circulatory conditions in at least the peripheral parts of the retina. This, however, does not imply that the central parts will not deteriorate any further.

Case 7.—J. B. complained of a considerable reduction in the visual acuity of both eyes. Ophthalmoscopic study revealed senile atrophy of the disks and macular and paramacular chorioretinal degenerations. The peripheral parts of the retina did not show marked abnormali-

ties. The electroretinograms of both eyes showed subnormal b-potentials (Fig. 7). No improvement could be obtained after a prolonged stay in the dark prior to the recording of the electroretinogram. Vasodilatory therapy, too, failed to raise the low b-potentials to normal values. (This in contrast to the reaction in Case 12 of the same group.) In my opinion, arteriosclerosis of the choriocapillaris must in this case have produced irreversible changes in the photoreceptor cells, which, in turn, produced subnormal b-potentials. Such changes cannot be observed in their full extension with the ophthalmoscope. The senile atrophy of the disk may have been responsible for the narrowing of the visual fields.

Case 9.—J. R. had a condition diagnosed as degeneration of the macular region resulting from changes in the choriocapillary layer in both eyes. Retinal and choroidal vessels in the paramacular area showed gross alterations of a sclerotic nature. Disseminated atrophy of the retina and the choroid in the posterior pole was observed; the peripheral parts of both retinas showed only minor abnormalities. The electroretinogram was extinguished (Fig. 8).

I consider the histories in the three cases of this group representative of different stages of the same disease. In each case marked alterations of various intensities in

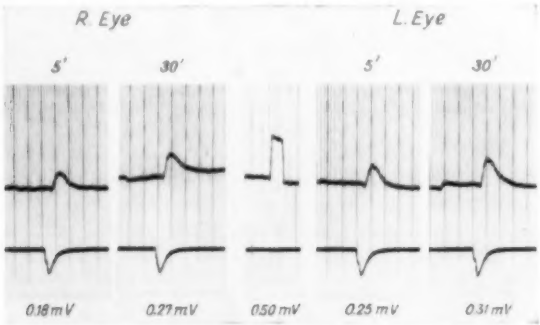

Fig. 5.—Influence of a prolonged rest in the dark on the b-potentials in a case of chorioretinal degeneration of the macula, resulting from sclerotic alterations in the choriocapillaris.

the choriocapillaris could be observed, while the retinal vessels outside the macular region showed no or, as in the last case discussed, only minor abnormalities.

I am inclined to believe that patients showing subnormal b-potentials with ophthalmoscopically normal paramacular retinas are suffering primarily from sclerotic changes in the choroid, which influence the metabolism of the photoreceptors.

Some patients with normal b-potentials and belonging to the group with chorioretinal degenerations, for example, P. P. (Case 6), also showing markedly arteriosclerotic and/or hypertensive retinal vessels. They belong, therefore, also to the group with degenerations accompanied by changes in the retinal vessels (Group 6) and will be discussed later.

There is no doubt that patients belonging to the groups discussed above (those with disciform and chorio-retinal degeneration) may show a normal electroretinogram because the sclerotic condition is really limited to the macular area, although, in my opinion, this is seldom the case. While use of the ophthalmoscope is generally limited to detecting localized lesions in the macular area, electroretinography is in most cases capable of revealing a more widespread alteration, which can be confirmed by the pathological examination.[3]

GROUP 3.—*Senile Cystoid Degeneration.*

CASE 2.—K. O. had complained of blurred vision in the left eye for one year, and vision in the right eye had begun to deteriorate during the preceding few weeks. The condition was diagnosed as cystoid degeneration of the macula in the left eye and chorioretinal degeneration in the

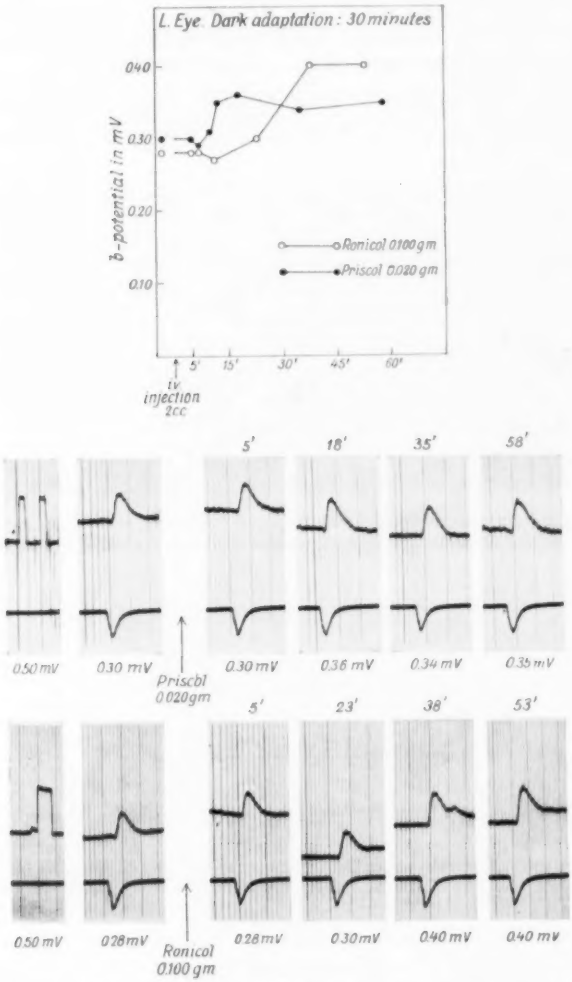

Fig. 6.—Further improvement in the b-potentials of the patient represented in Figure 5, following intravenous vasodilation after 30 minutes of rest in dark. Effect of Roniacol, 0.100 gm., is superior to that of tolazoline, 0.020 gm.

right eye. The electroretinogram of the right eye was of the subnormal type, typical of a sclerotic process in the macular region (Group 2), whereas the more affected (left) eye showed a normal b-potential. Figure 9 illustrates the marked discrepancy between the visual acuity and the data obtained in the electroretinographic examinations. The electroretinogram points to more widespread involvement of the right eye, though its visual acuity was relatively good. The electroretinogram recorded several months later showed exactly the same pattern.

Case 4.—A. J.'s left eye presented a picture typical of cystoid degeneration of the macula lutea. No abnormalities were found in the right eye. The electroretinograms of both eyes were of the subnormal type. However, a prolonged rest in the dark improved the b-potentials considerably: The left eye, after the usual dark adaptation of five minutes, gave a subnormal electroretinogram, with a b-potential of 0.17 mv., which, after 30 minutes in the dark, increased to a normal record, with a b-potential of 0.28 mv. The right eye after five minutes of dark adaptation gave a subnormal electroretinogram, with a b-potential of 0.15 mv., and after 30 minutes of dark adaptation a normal electroretinogram, with a b-potential of 0.30 mv.

In the cystoid degenerations of the macula the normal electroretinogram prevails. As a possible cause of this affection, the sclerotic changes in the choriocapil-

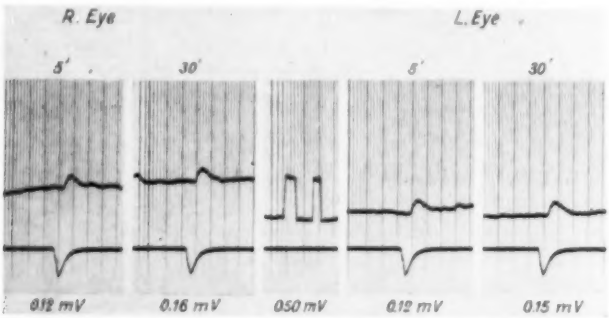

Fig. 7.—Insignificant improvement in height of the b-potential in a patient with bilateral senile chorioretinal degeneration following a prolonged rest in the dark. Compare with Fig. 5.

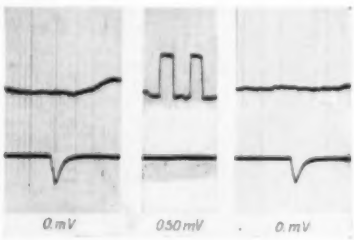

Fig. 8.—Extinguished electroretinograms in a case of chorioretinal degeneration of the macula with marked sclerosis of the choroidal vessels in posterior pole of the eyes.

laris are less important than in the disciform and chorioretinal forms of macular degeneration. The importance of sclerosis of the retinal vessels, however, must be stressed. A further evaluation will be given.

Group 4.—*Senile Colloid Degeneration.*—The data do not present material for discussion.

Group 5.—*Senile Circinate Degeneration.*

Case 2.—The history of G. B. is of interest, as the subnormal electroretinogram of the right eye pointed, in my opinion, to sclerosis of the choriocapillaris. One year later, the fundus picture had changed considerably, showing a marked disciform degeneration of the macular area of the affected eye. As I pointed out before, the disciform macular degeneration bears a special

relation to the sclerosis of the choriocapillary layers. In all the other cases belonging to the circinate group, the electroretinogram was normal, or even supernormal. Consideration of the latter type of electroretinogram will be included in the discussion of the following group.

GROUP 6.—*Senile Degeneration of the Macula Accompanied by Minor Signs of Retinal Arteriosclerosis and/or Hypertension.*—Excluded are cases in which ophthalmoscopy revealed pronounced retinopathy.

CASE 12.—The right eye showed degeneration of the macula with signs of some arteriosclerosis of the retinal vessels, namely, irregularity of the lumen and enlargement and tortuosity of the vessels. No sclerotic changes in the choroidal vessels could be observed. The left eye showed the same pattern in the vessels, without, however, any alteration in the macular region. General examination revealed marked arteriosclerosis, without hypertension. Electroretinography gave a negative+ electroretinogram, with normal b-potentials of 0.32 and 0.34 mv. (Fig. 10*A*). The same pattern was found 20 months later; only a slight increase in the b-potentials could be recorded after 30 minutes of rest and dark adaptation (Fig. 10*B*). The ophthalmic picture, however, had changed, since it showed pronounced disciform degeneration of the right macula and minor changes in the left one. The retinal vessels had the same appearance as before.

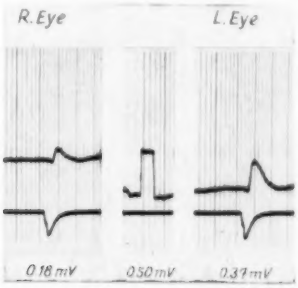

Fig. 9.—Subnormal electroretinogram in the right eye of a patient with chorioretinal degeneration with slight impairment of visual acuity (visual acuity in right eye, 6/10), combined with a normal electroretinogram in the left eye, showing a cystoid degeneration of the macula with slight symptoms of arteriosclerosis of the retinal vessels (visual acuity in left eye, 1/10). There is marked disagreement between the data obtained in electroretinography and the ophthalmoscopic picture.

The present case is an unusual instance of disciform degeneration with a negative + electroretinogram. In my opinion, the explanation for this behavior is that the sclerotic changes in the retinal vessels influenced the metabolism of the retinal neurones, especially those of the inner nuclear layer, from whence the b-wave is believed to originate. The changes in the choriocapillaris which are held responsible for the subnormal b-potentials, through an influence on the first neurone—the photoreceptors—were probably in this case less important.

In this connection, the electroretinographic data found in Case 1 of Group 1, are of interest. A disciform degeneration was diagnosed, the electroretinogram showing relatively high b-potentials and prominent a-waves (negative electroretinogram). In this case, too, sclerosis of the retinal vessels was observed.

In other cases belonging to the various groups of macular degeneration the same variation in the electroretinogram appeared: normal, supernormal, or negative+, all with some signs of sclerosis of the retinal vessels; irregularity of the lumina, and enlargement and tortuosity of the vessels in all. In all these cases

the condition of the second neurone was decisive, in my opinion, in determining the electroretinogram recorded. Supernormal b-potentials are, I believe, the result of increased irritability of the neurone of the second order, which could be due to a subnormal oxygen supply following the sclerotic changes in the retinal vessels. In a more advanced stage of the disease the supernormal reaction may turn into a subnormal one.

GROUP 7.—*Unclassified Cases of Senile Degeneration.*—As I pointed out earlier, it is impossible to classify every case satisfactorily. The group of unclassified cases shows, therefore, features of all the groups mentioned above.

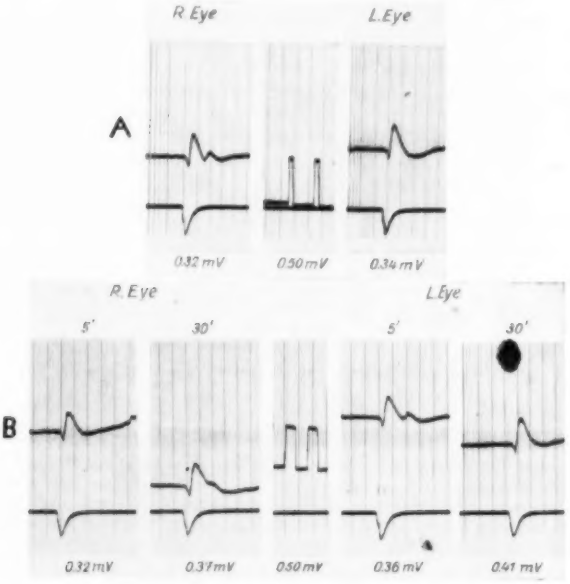

Fig. 10.—*A*, negative+ electroretinogram in a case of senile macular degeneration with minor signs of arteriosclerosis of the retinal vessels. *B*, electroretinograms of both eyes of the patient represented in *A*, recorded 20 months later, showing the same pattern, a slight increase in the b-potentials following a 30-minute rest in the dark.

COMMENT

As the electroretinogram is considered to be the total response of the whole retina following illumination, it is logical to expect a normal electroretinogram in those cases of macular degeneration in which the periphery of the retina does not show gross alterations. In this connection, Karpe's [4] statement is of importance.

> Small localized injuries to the retina do not have any effect on the electroretinogram if the rest of the retina is intact. It is therefore—in cases of cataract—not possible to diagnose senile degeneration of the macula by means of the electroretinogram.

From this it is logical to assume that Karpe [5] recorded a normal electroretinogram in a case of heredodegeneration of the macula. François,[6] in 22 cases of "macular degeneration," also found a normal electrical response. However,

my results are different, at least as concerns the senile degeneration of the macular area. On a previous occasion [2c] I demonstrated the normalizing effect of vasodilator therapy on the subnormal electroretinogram of a patient with senile chorioretinal degeneration. (Case 12, Group 2).

My results indicate that in cases of senile degeneration of the macula different types of electroretinograms can be found. In all the cases discussed above, ophthalmoscopically only the macular region appeared to be affected, but the pathological electroretinogram indicated a general retinal and/or choroidal involvement. The Table shows that most of the lower b-potentials were found in the groups of senile disciform degeneration and senile chorioretinal degeneration.

In discussing the electroretinogram above the age of 50, Karpe [7] gives 0.30 mv. as a normal value for the b-potential for men and 0.34 mv. for women. However, the b-potential decreases with advancing age, since his mean value for the b-potential between the ages of 60 and 64 is 0.31 mv., of 65 and 69, 0.25 mv., and of 70 and 74, 0.23 mv. It should be mentioned, however, that the last value refers only to three ophthalmoscopically normal persons showing no signs of hypertension or pronounced arteriosclerosis. If the last value is accepted as normal for persons above the age of 70, the majority of the b-potentials of my patients of this age group who had senile degeneration of the disciform or the chorioretinal type, fit in well. My data pertaining to the cases belonging to the other groups, however, are not at all in accordance with these "normal" values. Other points of disagreement with respect to the commonly accepted view that the electroretinogram is not affected by such localized macular processes as the senile degeneration of the macula is assumed to be are (*a*) the marked abnormality in the electrical response of one retina, or the significant differences between the electroretinograms of the two eyes, in cases in which the eyes show more or less the same pathological picture with the ophthalmoscope and deterioration of vision, and (*b*) the marked abnormality in the electroretinogram of the ophthalmoscopically normal eye when the disease seems to be unilateral.

It is reasonable to believe that in cases of disciform and of chorioretinal degeneration the sclerotic changes in the choroid as a whole are responsible for the subnormal b-potential, through an influence on the metabolism of the photoreceptors. The pathological findings, as given in the literature, confirm the concept of a primary lesion of the choriocapillaris. With the ophthalmoscope, however, only the macular lesion can be observed. In the groups showing normal, or even supernormal b-potentials, especially the group of senile degeneration accompanied by minor arteriosclerotic-hypertensive signs of the retinal vessels, the sclerotic changes in the choriocapillaris are believed to be of less importance. Here the influence on the metabolism of the second neurone must play a more important role, which influence may be exerted through sclerotic, and/or hypertensive changes in the retinal circulation. The increased irritability, with the resulting supernormal b-potential, may be a first sign of an insufficient retinal circulation.

A trend toward normalization of the supernormal, as well as of the subnormal, b-potentials during treatment tends to make the prognosis for the retinal condition as a whole rather favorable. The course of the macular process, and thus of the visual acuity, however, is in most cases not influenced by an improvement in the retinal condition.

SUMMARY

This article presents the results of a study of the electroretinograms in 80 cases of senile macular degeneration.

Subnormal b-potentials have been recorded in most cases of disciform degeneration of the macular area and of chorioretinal degeneration of Haab. Both types are characterized pathologically by sclerotic changes in the whole choriocapillaris.

While the ophthalmoscope could, on the whole, detect only local macular lesions, the electroretinogram revealed a more widespread sclerotic condition of the choriocapillary layers. The subnormal electroretinogram is assumed to be a result of interference with the normal metabolism of the photoreceptors.

The effect of vasodilator therapy in several cases could be observed by an increase of the b-potential.

Those forms of senile degeneration of the macula, which are associated with minor alterations in the retinal vessels of a sclerotic nature show mostly normal, supernormal, or negative + electroretinograms. A negative + or a supernormal electroretinogram is considered to be due to an increased irritability of the neurones of the second order. Normalization of the b-potential points to improvement of the circulation of the retina and/or choroid. Such an improvement, however, does not imply an improvement in the macular process.

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SPONTANEOUS HOLE IN ANTERIOR HYALOID MEMBRANE

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AFTER uncomplicated intracapsular lens extractions with or without an iridectomy, either complete or peripheral, it is very common to see a blob of vitreous in the anterior chamber. As long as the vitreous does not come into contact with Descemet's membrane, no untoward effects are to be expected.

As a rule, the slit-lamp microscope does not reveal any opening in the anterior hyaloid membrane through which the vitreous blob protrudes. Berliner [1] states:

> Following intracapsular cataract extraction, where herniation of the vitreous is commonly seen, rupture of the "hyaloid" and spilling of vitreous substance into the anterior chamber may occur spontaneously months postoperatively. This phenomenon may be attended by slight ciliary injection. Improvement in visual acuity has been noted following such an episode.

However, in no textbook on biomicroscopy of the eye is there an illustration of the lesion which I am reporting.

Mr. P. C., a white man aged 64, in December, 1950, had a nuclear cataract extracted from his left eye. The postoperative course was uneventful and corrected vision was 20/30. In January, 1953, the lens in the right eye, which also had pronounced nuclear sclerosis, was extracted intracapsularly, the Arruga forceps being used. Preplaced sutures were used. There were no postoperative complications, and at no time was there any untoward inflammatory reaction. About 10 weeks after operation, during routine examination, a very small hole was seen in the anterior hyaloid membrane somewhat temporal to and below the center. Strands of vitreous flowed toward the hole and out into the anterior chamber. No vitreous was in contact with Descemet's membrane. There were several whitish nodules in the vitreous strands, which extended toward the hole. There were no cells in the aqueous or deposits on Descemet's membrane, and the globe was not injected. The intraocular tension was 20 mm. Corrected vision was 20/30. No change has taken place in the ocular status.

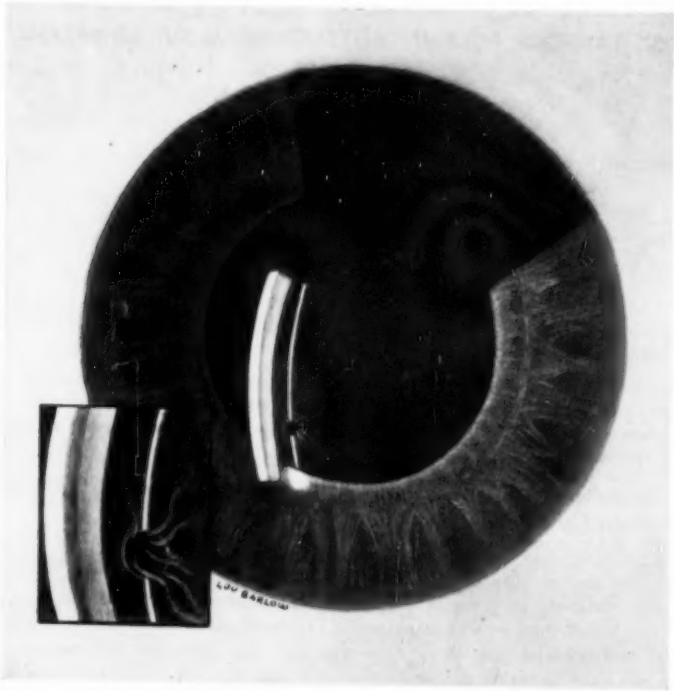
I would postulate that the site of the attachment of the hyalolenticular membrane is at the precise point where the hole developed. In removing the lens by the tumbling method, enough traction was made at this point to tear a minute opening in the anterior hyaloid membrane, through which strands of vitreous extruded. According to Berliner, however, the hole develops several weeks or months after the lens extraction. Why or how this happens I cannot explain, unless there is a temporary slight increase in the intraocular tension sufficient to produce a minute break in the hyaloid membrane at this precise point. Whatever the cause, such a tear is an extremely rare finding, but of no apparent clinical significance.

A recent report by Irvine [2] calls attention to this condition.

> Occasionally, at the first postoperative examination, a small hole is seen in the vitreous face. This probably represents the site in the patellar fossa where the anterior hyaloid was slightly

adherent to the lens and from which it pulled away during delivery. This site is the thinnest part of the hyaloid and a small hole often develops here during the postoperative period.

When a hole is seen, the rest of the vitreous face is usually behind the pupil, the hole formation seeming to lessen the tendency for vitreous to herniate into the anterior chamber. There are many instances in which there is development of a hole and later, through this hole, there is streaming of free vitreous into the anterior chamber.



Hole in the anterior hyaloid membrane as seen with the slit-lamp microscope.

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MANDIBULOFACIAL DYSOSTOSIS

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MANDIBULOFACIAL dysostosis, a rare congenital malformation of the facial bones, was first described in 1889 by Berry,[1] who presented two cases. Treacher Collins [2] presented two additional cases in 1900, at which time the condition became known as the "Treacher Collins' syndrome." Since then, the various titles under which the syndrome has been presented have served only to confuse its recognition. Fischer [3] described it as "congenital craniofacial hemiatrophy"; Sanvenero-Roselli * called it "microgenia with bilateral microtia and congenital malformation of the palpebral apparatus"; McEnery and Brennemann [6] named it "multiple facial anomalies," and Debusmann [7] labeled it "familial combined facial malformation in the region of the first visceral arch."

Franceschetti,† in an attempt to clarify the nomenclature, designated the condition as "Mandibulofacial dysostosis." Contrariwise, this resulted in its becoming known as "Franceschetti's disease."

A detailed search of all available literature reveals only 38 reported cases, with but few from this country. Therefore, a new case of mandibulofacial dysostosis, with previously unreported pathologic changes, is herewith presented, together with a review of the features of the syndrome.

The chief symptoms of the syndrome as collated by Franceschetti and Klein,[10] consist of (1) palpebral fissures which slope laterally downward (antimongoloid) with colobomata in the outer portions of the lower lids, and occasionally, in the upper lids; (2) hypoplasia, principally of the malar bones and mandible; (3) malformation of the external ear and, occasionally, of the middle and inner ear; (4) macrostomia, high palate, and irregular disposition of the teeth, with malocclusion; (5) blind fistulae between the angles of the mouth and ears; (6) atypical hair growth, in the form of tongue-shaped processes of the hair line extending toward the cheeks, and (7) facial clefts and skeletal deformities, such as missing thumbs.

Other, but less salient, features may be ocular (absence of the lashes in the internal part of the lower lid, hypoplasia of the orbits, microphthalmos, cataract, atresia of the lacrimal canals, ectopia of the pupils, convergent strabismus, and a double row of lashes on the lower lids); nasal (enlarged frontal sinuses, absence of the frontonasal angle, partial atresia of the nostrils or nasal fossa, and rudimentary sphenoid sinus); oral (cleft palate, large mouth, and underdeveloped epiglottis); aural (absence of external auditory canal, hypoplasia of auricles, and absence of mastoid cells), and bony defects of skull (absent or rudimentary zygomatic arches,

From the Chicago Medical School and Mount Sinai Hospital.

* References 4 and 5.

† References 8 and 9.

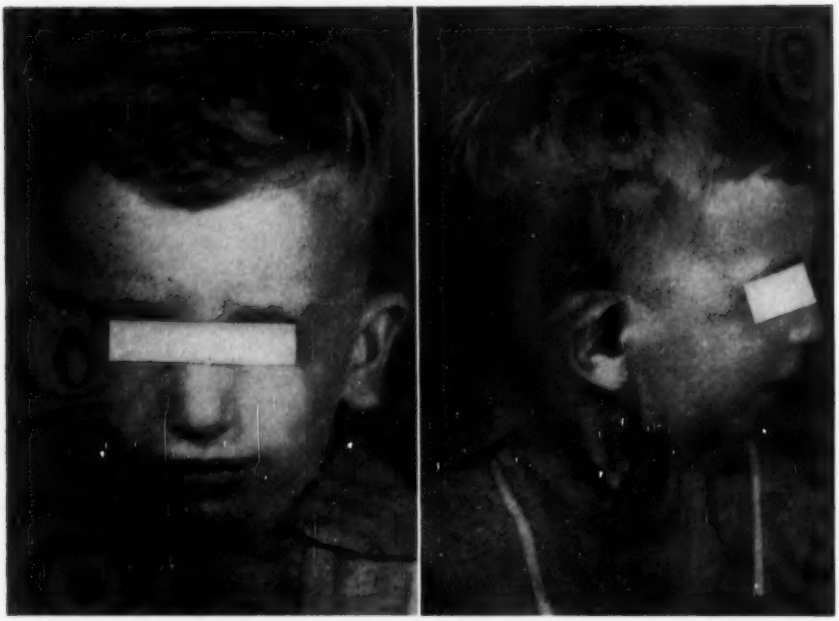

Fig. 1.—Mandibulofacial dysostosis. Shown are malformation of ears, mandibular hypoplasia, and fish-like face.

Fig. 2.—Mandibulofacial dysostosis. Shown are absence of zygomatic arches, deformity of inferior orbital margins, and irregular dentition.

micrognathia, fish-like or bird-like face, receding chin, temporomandibular malarticulation, narrow and deep sella turcica, hypoplasia of the petrous bone, enlarged sphenoidal and sphenomaxillary fissures, and prognathism).

Additional features may include agnesia of the frontalis muscle, mental retardation, harelip, clubfoot, and synostosis of joints.

Five forms of the disease can be classified.

1. Complete

2. Incomplete, in which the deformity is less marked. The ears may be almost normal, but usually deafness is present

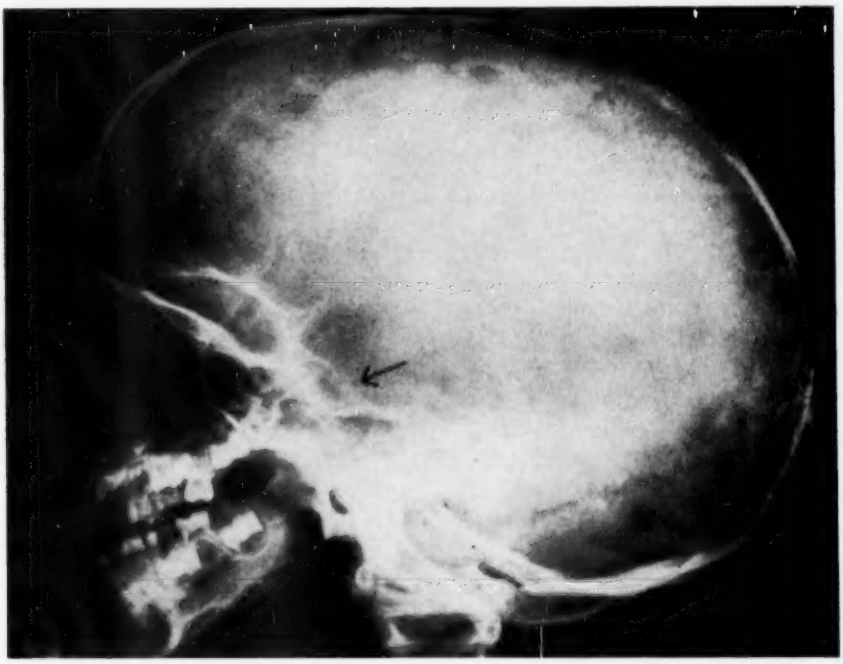

Fig. 3.—Mandibulofacial dysostosis. Shown are widening of the sella, accentuated digital impressions, and dolichocephaly.

3. Abortive, in which only the lid anomalies exist

4. Unilateral, with hypoplasia of the muscles of the face and other symptoms on one side

5. Atypical

The hereditary aspect of the condition has been proved by its appearance in several generations. There is an irregular dominant form of transmission. Debusmann cited nine typical cases in three generations. Straith and Lewis [11] presented a mother and four children with mandibulofacial dysostosis.

The embryonic aberration and pathogenesis are described by Ida Mann.[12] Ostensibly, the condition evolves from delayed ossification of the mesodermally

derived facial bones of the first visceral arch. This deficiency occurs about the seventh embryonic week. Consequently, this results in hypoplasia and malposition of the facial bones and tissues.

Mandibulofacial dysostosis must be differentiated from cleidocranial dysostosis, craniofacial dysostosis (Crouzon), and acrocephaly with syndactyly.

CASE PRESENTATION

The case presented here is that of a 5-year-old boy, whose hereditary history is without significance. The following features of mandibulofacial dysostosis were present: (1) antimongoloidpalpebral fissures with colobomata of the outer portions of the lower lids and absence of the lashes in the lateral parts of the lids; (2) hypoplasia of the malar bones, rudimentary zygomatic arches, micrognathia, and receding chin; (3) malformation of the external ear; (4) microstomia, high-arched palate, speech defect, irregular disposition of the teeth, and malocclusion; (5) blind fistulae between angles of the mouth and ears; (6) atypical hair growth with tongue-like extensions; (7) frontonasal angle diminished; (8) fish-like face, and (9) mental retardation.

This case is classified as one of the complete type. Features that have not been previously noted as associated with the syndrome are widening of the sella, accentuated digital impressions of the skull, dolichocephaly, and defective inferior orbital margins.

The treatment, which is in progress, consists of surgical plastic repair.

SUMMARY

A new case of mandibulofacial dysostosis, with a brief summary of the history and features of the syndrome is presented.

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RETROLENTAL FIBROPLASIA

Definitive Role of Oxygen Administration in Its Etiology

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CAMPBELL,[1] of Australia, in July, 1951, published observations on a series of cases of retrolental fibroplasia which suggested that the disease might be due to excessive administration of oxygen.* Since this time, Campbell's experiences have been shared by several investigators in different parts of the world: by Ryan,[3] in Australia; by Crosse and Evans,[4] in England; by Patz, Hoeck, and De La Cruz,[5] in the United States, and by Goldman and Tobler,[6] in Switzerland, to mention a few.

However, in December, 1951, and again in March, 1952, the opposing view was propounded by Szewczyk,† of St. Louis, namely that the disease was due to anoxia, and that oxygen not only could prevent the disease but would also be effective in its therapy. Szewczyk's views have been given some support by Ingalls [9] on the basis of laboratory studies; as a result of these directly opposing theories, there has existed confusion in the minds of those responsible for the care of premature babies.

This presentation lends support to the hyperoxygen theory of the etiology of retrolental fibroplasia. Weekly studies from birth of the fundi of 327 premature babies indicate that retrolental fibroplasia can indeed be largely prevented by curtailing the amount of oxygen given.

PROCEDURE AND RESULTS

The studies to be reported were carried out, in 1951, in the nurseries of Presbyterian and Lincoln Hospitals, in New York city, and, since early in 1952, in the premature nurseries of Catherine Booth Mothers' Hospital and the maternity pavilion of the Royal Victoria Hospital, in Montreal.

In 1951, the babies under observation were receiving oxygen for relatively prolonged periods of time,‡ and the incidence of retrolental fibroplasia observed during this period was high. Table 1 shows that 60 of 160 babies developed acute manifestations of the disease, of whom 18 (or 30%) had severe forms.

The work done in New York was supported by a grant from the Dunlevy Milbank Foundation, Inc.

Presented at the Sixteenth Annual Meeting of the Canadian Ophthalmological Society, Minaki, Ont., June 15, 1953.

From the Institute of Ophthalmology of the Presbyterian Hospital, New York, and from the Department of Ophthalmology of the Royal Victoria Hospital.

* Prior to this, a possibly significant correlation had also been noted by Kinsey and Zacharias.[2]

† References 7 and 8.

‡ A significant number of these babies received continuous oxygen for more than 30 days after birth, and the majority received it for more than 10 days.[10] The initial rate of flow was generally about 6 liters per minute. A policy of gradual rather than sudden oxygen withdrawal was practiced. Incubators in use were of the Isolette and the Gordon-Armstrong types.

During the first five months of observation in Montreal (February to July, 1952), no conscious effort was being made to limit the amount of oxygen, but this was generally less than that being given in New York. Table 2 shows the incidence of the disease during this period. In the last column of the Table is calculated the number of cases expected in each birth weight group on the basis of 1951 experiences. In 43 babies observed, the actual number of cases (6) was less than the expected number (10), but this difference cannot be considered significant.

TABLE 1.—*Incidence of Retrolental Fibroplasia Observed in New York in 1951*

Birth Weight, Lb.	No. of Babies	No. of Cases	Incidence, %
3 and less	62	36	58
3-3½	43	14	33
3½-4	42	8	19
4-4½	13	2	15
Total	160	60	

TABLE 2.—*Incidence of Retrolental Fibroplasia Observed in Montreal, February to July, 1952*

Birth Weight, Lb.	No. of Babies	No. of Cases Observed	No. of Cases Expected on Basis of 1951 Experience
3 and less	5	2	2.9 (58% of 5)*
3-3½	8	3	2.6 (33% of 8)
3½-4	13	1	2.5 (19% of 13)
4-4½	17	0	2.5 (15% of 17)
Total	43	6	10.5

* Table 1 shows origin of percentage figures.

TABLE 3.—*Incidence of Retrolental Fibroplasia Observed in Montreal, August, 1952, to October, 1953*

Birth Weight, Lb.	No. of Babies	No. of Cases Observed	No. of Cases Expected on Basis of 1951 Experience
3 and less	24	2	13.9 (58% of 24)*
3-3½	17	0	5.6 (33% of 17)
3½-4	37	0	7.0 (19% of 37)
4-4½	46	0	6.0 (13% of 46)
Total	124	2	32.5

* Table 1 shows origin of percentage figures.

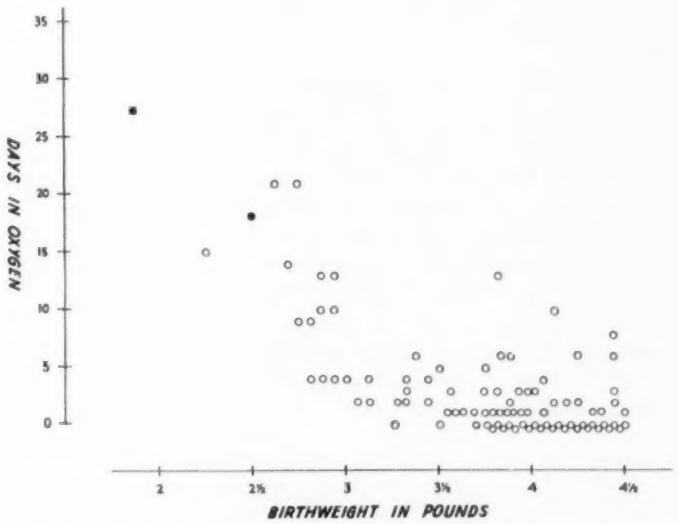
In July, 1952, and subsequently, the oxygen being administered was reduced to minimal amounts,§ in most cases to an amount just sufficient to prevent cyanosis. An extremely low incidence of cases occurred, with a complete absence of severe forms.

§ The Chart shows that none of these babies received oxygen for more than 30 days after birth, and that the majority received it for less than 10 days. Where the rate of flow was initially 6 liters per minute, it was soon reduced. Other babies received 4, or even 2, liters per minute from the beginning, while still others received no oxygen at all. Oxygen withdrawal was gradual, never sudden. In one nursery the Isolette and Gordon-Armstrong types of incubator were in use, and in the other nursery, the Castle Humidicrib. Oxygen concentrations within these incubators were tested by means of the Beckman oxygen analyzer.

TABLE 4.—*Duration, in Days, of Continuous Oxygen Received by Babies in Montreal, Who Developed Retrolental Fibroplasia and by Babies Who Were Not Affected* **

Birth Weights, Lb.			
3 and Less	3-3½	3½-4	
(28)	(32)	(14)	13
21	(28)	(7)	7
(21)	24	6	6
21	(10)	5	5
(18)	6	3	3
(18)	5	3	3
(18)	(5)	3	3
17	5	3	3
17	4	3	1
15	4	1	1
14	4		
13	3	1	1
10	3	1	1
10	2	1	1
9	2	1	1
9	2	1	1
4	2	1	1
4	2	1	0
4	2	0	0
4	1	0	0
4	0	0	0
2	0		

* Each figure in this table shows the number of days that an individual baby received continuous oxygen from birth. The parentheses are values for the babies who developed retrolental fibroplasia. The horizontal line marks the median of each group.



Duration, in days, of continuous oxygen received from birth by babies observed in Montreal from August, 1952, to May, 1953. The solid circles represent babies who developed retrolental fibroplasia; the hollow circles, babies who were not affected.

Table 3 shows that of 124 babies followed, only 2 developed manifestations of retrolental fibroplasia. These both had mild forms, which did not advance beyond Stage 1 (classification of Reese and co-workers [11]). They were two of our smallest babies, weighing 2½ and 1¾ lb. (1,134 and 794 gm.), respectively, at birth, and they received relatively larger amounts of oxygen than others in the same birth weight group (for 18 and 28 days, respectively). Both showed complete regression, and in a retrospective study would have been declared normal.

On the basis of 1951 experiences, the expected number of cases in a population with this birth weight distribution would be 32 (calculated in the last column of Table 3), and of these, 30% (or 10 cases) would be severe forms. Applying the chi-square formula to these figures, P is found to be less than 0.001; that is, there would be less than 1 chance in 1,000 that the difference could be due to chance alone.

This in itself, however, does not necessarily prove a causal relationship between oxygen administration and retrolental fibroplasia. Some other causative factor could conceivably have been operating at the same time, even though no other significant difference could be noted in the management of the babies in New York and Montreal. To establish a more certain relationship, it is necessary to compare the amount of oxygen received by babies developing the disease with that received by those not affected.

This was done in Table 4, which includes all cases seen in Montreal from February, 1952, until June, 1953, on whom there are accurate oxygen data.‖ In each birth weight group, the same phenomenon is seen, namely, the babies who developed retrolental fibroplasia received oxygen, on the whole, for significantly longer periods than those who did not.¶ When the t test is applied to these figures, P is found to be less than 0.001; that is, there is less than 1 chance in 1,000 that this could be a chance distribution.

COMMENT

The data presented in this paper show (1) that when the oxygen being administered routinely to premature babies was reduced to minimal amounts (in most cases to amounts just necessary to prevent cyanosis), there was a striking fall in the incidence of retrolental fibroplasia, for which no other explanation could be found, and (2) that babies who developed the disease received oxygen on the whole for significantly longer periods than those who did not. Both these relationships were found to have a high degree of statistical significance.

The findings, therefore, support the already published observations of Kinsey and Zacharias,[2] Campbell,[1] Ryan,[3] Crosse and Evans,[4] Patz and co-workers,[5] Goldman and Tobler,[6] and others who favor the hyperoxygenation theory of the etiology of retrolental fibroplasia.

‖ Included in the Table are two additional cases from St. Mary's Hospital and one from the Children's Memorial Hospital, Montreal. Omitted from the Table are a few normal babies in the February-July, 1952, group, on whom exact data regarding oxygen are not available.

¶ That this is not an absolute relationship, however, is also readily seen. For example, in the 3 to 3½ lb. (1,307 to 1,534 gm.) birth weight group, a baby who received continuous oxygen for five days after birth developed the disease, while another baby who received it for 24 days did not.

The figures also suggest that in the lower birth weight groups somewhat longer periods of oxygen administration might be more safely tolerated than in the higher birth weight groups.

They place us at variance with those who support the anoxia theory,# or any middle-of-the-road theory,[12] or with those who advocate the use of oxygen in the therapy of the disease.*

While an alternate case study would have been preferable, moral considerations prohibited this method of study.

Szewczyk,[8] while also noting (in 1951) that the babies who developed retrolental fibroplasia were kept in an oxygen incubator on an average longer than those who did not develop the disease (30 days, as compared with 20 days), argued conversely:

> If we consider the usual pediatric criteria which are used to determine when a child is to be placed in a normal atmosphere, it becomes immediately apparent that the group of children who developed retrolental fibroplasia were actually more oxygen deficient than the others.

This argument does not hold for the group of babies reported here. In 1951, our babies were for the most part given oxygen above and beyond their needs, and the incidence of the disease was high. In 1952, when the oxygen was reduced to minimal amounts, and oxygen deficiency made more likely, the incidence of the disease declined.

Our experiences also fail to support the theory that the disease is due to a relative anoxia induced by too rapid withdrawal from high oxygen concentrations, since in this series slow weaning from oxygen was the rule and cases even developed during the course of oxygen administration. Our studies, rather, support the concept that prolonged high oxygen concentration is injurious as a result of a direct effect. Whether rapid removal from high oxygen can *increase* the tendency toward retrolental fibroplasia cannot be deduced from this study.

It has been suggested[13] that if the administration of oxygen were a causative factor in retrolental fibroplasia, there should be a correlation between the use of oxygen and the date of onset of the disease. However, Silverman and co-workers[10] have shown that the age of onset of the disease is related, rather, to the birth weight of the baby. They noted an inverse relationship, the disease having its onset late in small infants and early in large infants. This observation suggests that the exact moment of onset of morphological changes is dependent on the stage of embryonic development of the tissues concerned more than on the date of cessation of any extraneous etiological agent.

A high blood oxygen saturation in the early days of postnatal life may be the stimulus needed to initiate the morphological changes. The exact time of onset of these changes, however, may depend on a tissue susceptibility present during a limited period of the developmental cycle.

Cases of retrolental fibroplasia have been reported in babies to whom no oxygen has been administered. This is not incompatible with the hyperoxygen theory, since

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References 7, 8, and 9.

* In the series reported here, severe forms have been observed occurring in babies who had never been removed from an oxygen-enriched atmosphere. In June, 1952, a baby who showed the first signs of the disease shortly after his removal from oxygen was placed back in oxygen when the disease appeared to be progressing. The disease nevertheless progressed to Stage 5 (classification of Reese and co-workers). Szewczyk, references 7 and 8.

the oxygen saturation of arterial blood in utero is approximately 50%,[14] and after birth rises in a few hours to approximately 90% in room atmosphere. Addition of further oxygen may merely serve to accentuate this difference.

In a disease such as retrolental fibroplasia, with a high rate of spontaneous regression, and with an incidence that has been reported to vary from time to time in the same nursery, one must be cautious in accepting any method purporting to prevent the disease or reduce its severity. In the hyperoxygen theory, however, we have for the first time a theory which finds support from several different observers in different parts of the world. It would seem advisable at this time, therefore, to urge the administration of only minimal amounts of oxygen to premature babies until stronger evidence to the contrary can be presented.

SUMMARY AND CONCLUSIONS

In a comparative study of 327 premature babies, a correlation between the incidence of retrolental fibroplasia and the duration of continuous oxygen therapy has been demonstrated.

When the oxygen being administered routinely to premature babies was reduced to minimal amounts, there was a striking fall in the incidence of this disease. It occurred in only 2 out of 124 babies, and both cases were mild. On the basis of previous experience, 32 cases would have been expected to occur in a population with this birth weight distribution.

Babies developing the disease received, on the whole, significantly more oxygen than those who did not develop it, although the relationship is not an absolute one.

The disease would seem to be directly related to hyperoxia, rather than to any secondary anoxia induced by oxygen withdrawal. Whether rapid removal from high oxygen can *increase* the tendency toward retrolental fibroplasia cannot be deduced from this study.

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UVEITIS IN CHILDREN

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UVEITIS in children, though relatively uncommon, may be a very distressing problem. The literature on the subject is scant and for the most part incidental to reports concerned with toxoplasmosis,* sarcoidosis,[6] or juvenile rheumatoid arthritis (Still's disease).† The only series of children with uveitis was reported by Thomson [13] (1928), whose 10 cases had in common a chronic course, uveal tract disturbance without visible fundus change or iritis, and unilateral involvement; apparently the etiology was unknown. Ravin [14] (1944) described a case of iritis following measles in a 3-year-old girl; Hallett [15] reported a case of iridocyclitis coincident with chickenpox in a 7-year-old boy, and Wilder [16] (1950) reported the finding of nematode larvae in the granulomatous lesions of a series of eyes enucleated from children.

The purpose of the present study was to analyze all the cases of childhood uveitis seen in connection with the uveitis survey being conducted by the staff of the Francis I. Proctor Foundation for Research in Ophthalmology and the Department of Ophthalmology, University of California Medical Center. A total of 810 cases of uveitis have been surveyed in a five-and-a-half-year period, and 47 of them, or 5.8%, were in patients under 16 years of age. This figure is unquestionably higher than the general average which is probably closer to 2 or 3%. Blegvad [17] (1941) found in Denmark that 2.2% of 896 cases of uveitis were in children under 15 years of age. The higher percentage in our series was probably due to the special interest of one of us (M. J. H.) in toxoplasmosis; many cases of healed chorioretinitis were referred for study which might otherwise have been ignored.

The records of all cases were drawn from the files of the uveitis clinic, to which both clinic and private patients are referred. They were studied as outpatients or hospital patients, and each was examined by at least one of us. Since many cases were referred because of suspected toxoplasmosis, and most other cases for diagnostic opinion because of recurrence, unusually severe, and/or special therapeutic problems, the series must be regarded as highly selected and unsuited to statistical

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* References 1, 2, 3, 4, and 5.

† References 7, 8, 9, 10, 11, and 12.

analysis. Our evaluation of certain survey procedures changed during the five and a half years, moreover, so that some of the tests applied early in the study were discarded and others were initiated.

All the records contained the following data: (1) brief medical history and detailed ophthalmic history; (2) complete physical examination; (3) detailed examination of the eyes, including slit-lamp biomicroscopy and funduscopy with pupils dilated; (4) skin tests, including the tuberculin, Frei, toxoplasmin, coccidioidin, histoplasmin, and Brucellergin tests; (5) serologic tests, including the Toxoplasma dye test, the Toxoplasma complement fixation test, Brucella agglutination and complement fixation tests, Q-fever complement fixation test, and the Kline and Kahn test for syphilis; (6) neutralization test for toxoplasmosis; (7) white blood cell count and differential count; (8) sedimentation rate, and (9) roentgen chest examination

TABLE 1.—*Etiologic Diagnosis or Disease Association in Anterior Uveitis (Eighteen Cases)*

Diagnosis or Association	No. of Cases	Age at Onset, Yr.
Heterochromic iridocyclitis	1	13
Boeck's sarcoidosis	1	11
Syphilitic keratouveitis	1	6
Traumatic iritis	1	8
Brucella keratouveitis	1	14
Juvenile rheumatoid iritis	1	1½
Unknown	12	

TABLE 2.—*Types of Anterior Uveitis of Unknown Etiology (Twelve Cases)*

Type	No. of Cases
Iridocyclitis, nongranulomatous	5
Iridocyclitis, granulomatous	3
Cyclitis	4

and special films of joints, skull, and phalanges when indicated by the history or physical examination.

The patient's age at the onset of the uveitis was determined whenever possible. In many of the cases with healed chorioretinitis it was impossible to state when the lesions had first occurred. In cases in which there was a healed macular lesion with a squint it seemed logical to assume that the lesion had been present at birth or soon thereafter.

ANALYSIS OF CASES

There were 47 children under 16 years of age in our total series of 810 cases of uveitis. Their cases could be divided into those of anterior and those of posterior uveitis, depending on which portion of the uvea was involved primarily, and there were more than half again as many cases of posterior uveitis (29) as there were cases of anterior uveitis (18). Curiously enough, there were no instances of generalized uveitis among the children.

Anterior Uveitis (18 cases).—An etiologic diagnosis, definite or presumptive, or a specific disease association was determined in 6 of the 18 cases of anterior uveitis. The 12 cases in which we were unable to determine the etiology or any specific disease association could be typed as shown in Table 2. The findings in

these 12 cases are elaborated in Tables 3, 4, and 5. Table 3 shows that four of the five cases of nongranulomatous iridocyclitis were acute and that the fifth was subacute. Three of the five patients had definite allergic diatheses. In one of these (a 12-year-old girl, M. M.) the eyes cleared simultaneously with the removal of a pet cat. Table 4 shows that the three cases of granulomatous iridocyclitis were chronic. One of the patients (R. S.) had one painful eye enucleated, and the histological studies showed only a nonspecific type of chronic inflammation. Cultural studies were not made. The four cases summarized in Table 5 were listed as instan-

TABLE 3.—*Iridocyclitis, Nongranulomatous Type (Five Cases)*

Patient	Sex	Age at Onset, Yr.	Eye(s) Involved	Skin Test and Serology	Positive Findings
D. C.	M	6	O. U.	—	Acute iridocyclitis; allergic diathesis
B. K.	F	5	O. U.	—	Acute iridocyclitis; allergic diathesis
J. W.	M	10	O. U.	—	Acute iridocyclitis, recurrent
A. E.	M	10	O. U.	—	Subacute iridocyclitis
M. M.	F	12	O. U.	—	Acute iridocyclitis; allergic diathesis

TABLE 4.—*Iridocyclitis, Granulomatous Type (Three Cases)*

Patient	Sex	Age at Onset, Yr.	Eye(s) Involved	Skin Test and Serology	Positive Findings
R. S.	F	7	O. U.	—	Chronic iridocyclitis; band keratitis
M. D.	F	14	O. D.	—	Chronic iridocyclitis; allergic diathesis
P. B.	F	8	O. U.	—	Chronic iridocyclitis; eosinophilia, 2-19%

TABLE 5.—*Cyclitis (Four Cases)*

Patient	Sex	Age at Onset, Yr.	Eye(s) Involved	Skin Test and Serology	Positive Findings
L. H.	F	11	O. U.	—	Allergy; crystalline deposits on irises
W. N.	M	12	O. U.	Brucella +	Allergic diathesis
G. C.	M	9	O. U.	—	Eosinophilia, 7-10%
M. D.	M	4½	O. U.	1st PPD +	Chronic cyclitis; band keratitis

ces of cyclitis because there was very little sign of reaction in the anterior chamber and no visible chorioretinitis; since in some cases the extreme periphery of the fundus could not be seen, we cannot say that in these there was no peripheral chorioretinitis. Anterior vitreous opacities constituted the most interesting finding in this group. The irises of one patient (L. H.) with a chronic bilateral cyclitis showed crystalline deposits that were thought to be lipoidal in nature.

Posterior Uveitis (29 cases).—A presumptive diagnosis of toxoplasmosis was made in 10 of the 29 cases of chorioretinitis. The significant findings in these and in the 19 undiagnosed cases are summarized in Tables 6, 7, and 8. The chorioretinitis was active in only 7 of the 19 cases that could not be diagnosed etiologically; of the 12 healed cases, 3 were associated with strabismus.

Differential Diagnosis of Uveitis in Children.—In the course of this survey six children were referred to the uveitis clinic who did not in fact have uveitis. Diag-

nostic study showed four of them to have Coats's disease. The ocular involvement was unilateral in all four, and three of the four patients were boys. The disease was definitely exudative in three and hemorrhagic in the fourth. In one case that was

TABLE 6.—*Chorioretinitis Presumed to be Due to Toxoplasmosis (Ten Cases)*

Patient	Age, Yr.	Sex	Toxo Dye Test	Skin Neut. Test	Skull X-Ray	Squint	Mental Retardation	Fundus
S. A.	3	M	.....	+	+	ST	+	O. S. healed
N. H.	8	M	.....	±	+	ST	+	O. U. healed
S. J.	14	F	.....	+	+	XT	+	O. S. active
S. F.	11	F	.....	+	+	XT	—	O. U. healed
S. F.	11	F	.....	+	+	ST	—	O. U. healed
F. S.	2½	M	1:2,048	..	—	ST	?	O. U. healed
G. S.	2½	M	1:256	..	—	ST	?	O. U. healed
L. B.	16	F	—	—	+	ST	+	O. U. healed
M. F.	12	M	1:128	+	+	—	—	O. S. active
L. S.	7	F	1:64	..	+	ST	—	O. U. healed

TABLE 7.—*Active Chorioretinitis of Unknown Etiology (Seven Cases)*

Patient	Age at Onset, Yr.	Sex	Skin Tests	Toxoplasma Dye Test	Fundus Lesions
R. T.....	15?	M	All —	0; neut. +	O. U.; O. D. active
B. W.....	14?	F	All —	0; neut. —	O. U.; O. D. active
R. S.....	12?	M	All —	—	O. D., subacute, elevated
C. E.....	6?	F	All —	—	O. S., proliferated
C. P.....	5?	F	All —	1:16	O. U., O. D. active
S. B.....	15?	F	1st PPD ± coccidioidin +	—	O. U., peripheral
I. S.....	3?	M	Toxo +	1:64	O. S., acute

TABLE 8.—*Healed Chorioretinitis of Unknown Etiology*

Patient	Age, Yr.	Sex	Skin Tests	Toxo, Dye Test	Comments
Y. P.....	7	F	All —	—	.....
M. J.....	8	F	All —	1:8	.....
P. B.....	9	M	All —	1:8	.....
J. W.....	12	F	Toxo +	...	Skin neut. test +; toxo?
W. L.....	12	M	Toxo +	...	Skin neut. test —
F. F.....	9	M	Brucella +	1:128	Toxo?
C. M.....	12	F	Brucella and coccidioidin +	1:128	Toxo?
J. G.....	10	M	1st PPD +	—	.....
E. B.....	6	M	All —	1:16	.....
P. F.....	4	F	All —	1:128	XT, age 1½ yr.; toxo?
J. N.....	15	F	All —	—	ST, age 1½ yr.
M. S.....	14	F	Toxo and histo +	1:16	ST from birth; toxo?

followed for five years the process resulted in a retinal detachment with loss of vision. Certainly, the exudative type of Coats's disease, especially in its early stages, is a condition that must be differentiated from uveitis.

The other two cases that presented a problem in differential diagnosis were examples of heredomacular degeneration without central nervous system involvement. In both of them all tests were negative, and clinically there were no signs of inflammation. The vitreous was clear. In both the disease had developed insidiously, and the lesions were bilateral and symmetrical.

COMMENT

One-third of the cases of anterior uveitis in this series could be given a reasonably definite etiologic diagnosis or could be explained as a manifestation of a specific disease entity. A case of heterochromic iridocyclitis occurred in a 15-year-old girl who had first noted blurring in one eye when she was 13 years old. The iris of the involved eye was lighter in color; the lens showed an early posterior subcapsular cataract. Heterochromic iridocyclitis of Fuchs usually manifests itself early in adult life and is rare in children.

Boeck's sarcoidosis involving the eyes was seen in an 11-year-old Negro girl. Clinically the uveitis was a granulomatous iridocyclitis. X-ray films of the chest showed hilar shadows, and lymph node biopsy findings were consistent with sarcoidosis. This disease is rare in children. Oblath and Farber [18] (1950) reported a series of 40 cases seen here at the University of California Hospital and at the San Francisco Hospital. Among these only three were in patients 15 years of age or younger. In a series of 29 proved cases of sarcoidosis reported by Woods and Guyton [19] (1944), 15, or 52%, were complicated by uveitis, and 2 of the 15 were in patients 15 years of age or younger. Although a definite diagnosis of systemic sarcoidosis can be made on the basis of lymph node and skin biopsies, tuberculin test, etc., the diagnosis of ocular sarcoidosis can be presumptive only, since all cases of the disease do not involve the eye. According to Thornhill and Thornhill,[6] eye involvement is especially rare in children.

Syphilitic keratouveitis was the definite diagnosis of the eye condition of a six-year-old boy with congenital syphilis. Serum tests for syphilis were positive, and the clinical findings were characteristic.

Brucella keratouveitis was the presumptive diagnosis in the case of a male dairy worker who was born and raised on a dairy farm. Raw milk was the only type of milk he had drunk from infancy, and he gave a history of several bouts of unexplained fever associated with malaise. His uveitis apparently dated from his 14th year. The clinical examination of the eyes showed a chronic iridocyclitis with nummular keratitis and early band keratitis. The Brucella skin test was positive, but the blood agglutinin and complement fixation titers were negative. Woods and Guyton [19] mentioned two cases of chronic brucellosis with nummular keratitis.

A case of uveitis presumed to be one of juvenile rheumatoid iritis was seen in an 8-year-old girl who had had arthritis when she was 1½ years old. Clinically the facies was bird-like, and the eyes showed a very chronic, low-grade iridocyclitis, early band keratitis, and complicated cataract. In spite of the current absence of arthritis, a presumptive diagnosis of juvenile rheumatoid iritis seemed to be justified. Blegvad [17] stated that a patient can recover completely from the arthritic element in this syndrome, and Franceschetti and Bamatter [9] pointed out that the typical eye findings of low-grade iridocyclitis, complicated cataract, and band-shaped keratitis are important early manifestations of juvenile rheumatoid arthritis (Still's disease).

No etiologic diagnosis, presumptive or definite, could be made in 12 cases of anterior uveitis. In 7 of the 12 cases there was an allergic diathesis, and 2 other cases showed a constant eosinophilia. Skin tests and serologic tests were consistently negative except in two cases, one with a positive Brucella skin test but a negative serologic test, the other with a positive tuberculin test.

While it is well known that a number of acute febrile diseases, particularly varicella, influenza, measles, and epidemic parotitis, are occasionally followed by uveitis, especially during their convalescent periods, it is noteworthy that none of the cases in this series followed any of the childhood exanthemata.

The infrequency of iridocyclitis in children is unexplained. The recent report of Reinhardt and Li [20] (1953) on the production of arthritis in experimental animals by the injection of the growth factor of the anterior pituitary hormone suggests that iridocyclitis, too, may eventually be explained on an endocrinologic basis, since arthritis and iridocyclitis are closely related clinically.

Healed chorioretinitis is a not uncommon finding in children's fundi. By virtue of our interest in congenital toxoplasmosis, however, we have undoubtedly seen more of it than one would find in a random sampling of cases. A presumptive diagnosis of toxoplasmosis was made in 10 of our children. A positive methylene blue dye test (Sabin and Feldman) and a positive complement fixation test are reliable and essential serologic criteria for the diagnosis of toxoplasmosis. If the child has clinical manifestations of the disease and the serologic tests are confirmatory, the diagnosis may be considered definite. After the age of 5 years, however, the problem becomes much more difficult because of the marked decrease in the patient's reaction to the methylene blue dye test and the reduced titer of complement-fixing antibodies in his serum. In fact, an unequivocal diagnosis is usually impossible in the older age group. Children with toxoplasmosis are usually mentally retarded in addition to having poor vision, and this adds to the importance of making the diagnosis. It is especially vital to the parents, since the chances are very good that subsequent children would be toxoplasmosis-free.

Sabin and Feldman [21] (1949) showed that toxoplasmosis was not the only cause of chorioretinitis and cerebral calcification. They described a syndrome rarely seen in infancy that is characterized by extensive destruction of brain tissue with hydrocephalus, by diffuse cerebral calcification, and by choroidal lesions associated with vascular changes; it can be differentiated from toxoplasmosis only by serologic tests or histopathological examination. There were no cases of this syndrome in our series.

Of the 19 cases of chorioretinitis in which we were unable to arrive at any sort of etiologic diagnosis, only 7 were active. The tuberculin reaction was negative in all seven, and only two showed Toxoplasma dye test titers (1:16 and 1:64 respectively). The chorioretinal lesions were bilateral in all but one case and recurrent in three. The one unilateral case showed a large proliferative choroidal lesion in a 6-year-old girl with a constant eosinophilia of 10%. It suggested a parasitic involvement, but no parasites were found. Since Wilder's (1950) finding of nematodes in histopathological preparations from endophthalmitis, an effort has been made to find the parasites in suspected clinical cases. So far this effort has been unsuccessful.

There were 12 cases of healed chorioretinitis of unknown origin, 3 of them with an associated strabismus. In each of these three cases the squinting eye showed a healed macular lesion. In their analysis of 1,000 cases of strabismus, Granström and Magnusson [22] (1949) found 6 to be due to toxoplasmosis. Congenital toxoplasmic infection was suspected in 5 of our 12 undiagnosed cases, but the evidence was insufficient for definitive diagnosis.

Our series did not contain any case of posterior uveitis associated with acute rheumatic fever, although one of us (M. J. H.) has seen in this disease a number of cases of choroidal inflammations resembling miliary tuberculosis. Rudolph,[23] on the other hand, studied a series of 105 cases of rheumatic fever in an Army hospital and found no ocular involvement.

It will be noted that no case in this series could be ascribed definitely to tuberculosis in spite of the widespread belief, particularly among German ophthalmologists, that this infection is the major etiologic factor in chronic forms of uveitis. Thiel,[24] for example, in a statistical review of all cases of endogenous uveitis treated at the Frankfurt University Eye Clinic and Hospital during the years 1935 to 1952, concluded that approximately 75% were tuberculous in origin. It is true that tuberculosis has declined strikingly in Northern California in recent years, but other extrapulmonary manifestations of the disease, particularly phlyctenular keratoconjunctivitis, are still being seen with considerable frequency. It is possible that tuberculosis tends to spare the uveal tract in childhood. One of us (P. T.), in the course of studies conducted with M. H. Fritz on phlyctenulosis among Alaskan Indian and Eskimo children,‡ was struck by the absence of uveitis except as a complication of the keratitis of phlyctenulosis. This type of secondary uveitis is ordinarily transitory and disappears with subsidence of the corneal attack, although, very exceptionally after repeated flare-ups, it may lead to atrophy of the globe. In a personal communication, Dr. Fritz stated that he had not seen a typical case of endogenous uveitis in the course of his examination of more than 2,000 Alaskan native children, in spite of the fact that, according to the results of tuberculin testing and the high incidence of pulmonary and bone and joint tuberculosis, the children were almost universally infected.

Healed pigmented foci are frequently seen in otherwise normal fundi, and there is usually no history of inflammation. Perhaps many of these will prove to be due to a defect in development rather than to an inflammatory process. At present, however, we cannot state that any of the healed chorioretinal lesions we have seen were developmental defects.

SUMMARY AND CONCLUSIONS

In a series of 810 cases of uveitis surveyed at the Francis I. Proctor Foundation for Research in Ophthalmology and the Department of Ophthalmology, University of California Medical Center, 47, or 5.8%, were in children under 16 years of age. Of the 47 cases, 18 were primarily of anterior uveitis and 29 were primarily of posterior uveitis.

Only one-third of the cases of anterior uveitis could be given an etiologic diagnosis, presumptive or definitive, or the condition identified as a manifestation of

‡ References 25, 26, and 27.

a specific disease. Of the 12 undiagnosed cases, the only frequent findings were an allergic diathesis, in 7 cases, and a constant eosinophilia, in 2 others, suggesting that allergy may be an important factor.

Of the 29 cases of posterior uveitis, a presumptive diagnosis of toxoplasmosis was made in 10, and 19 could not be explained. The practical importance of determining the presence or absence of toxoplasmosis is emphasized.

The chorioretinitis was active in only 7 of the 19 cases of posterior uveitis which could not be diagnosed etiologically.

Four cases of Coats's disease and two cases of heredomacular degeneration were referred as instances of uveitis.

Iridocyclitis is extremely rare in children; chorioretinitis is relatively common.

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Registry of Interesting Cases

It is the purpose of this section to record cases on the basis of interest only. No complete review of the literature is attempted and no claim for priority is implied except when specifically stated. Publication of case reports is subject to the concurrence of the Editorial Board on the interesting aspects of the case. It is requested that the reports submitted be as brief as is consistent with inclusion of the pertinent facts, bear a title that is as specific as possible, be prefaced by a paragraph that states why the author feels the case is of interest and be accompanied with photographs whenever possible.

BERYLLIUM AND DELAYED CORNEAL HEALING

Report of a Case

THE FOLLOWING case is reported because of its rarity. The literature has innumerable reports on beryllium toxicity in general and a few references to the conjunctiva. None were found of cases involving the cornea. This was surprising to us. It would seem likely that the injury would be more frequent. We might surmise that other cases failed to be reported because of the rapid, uneventful healing.

REPORT OF CASE

Our patient was first seen on June 12, 1952, with a chief complaint of pain in his left eye. An hour before arriving at the office, he broke a fluorescent bulb while attempting to remove it. He was looking up at the bulb, and fragments of the glass fell in his left eye. He experienced pain and a foreign body sensation in his eye. He came directly to our office.

Ophthalmological examination of the left eye revea'ed that the conjunctiva was injected and had a small laceration on the temporal side. The cornea had multiple epithelial abrasions with a small particle of glass imbedded at 3 o'clock near the limbus. Visual acuity was 20/30 in each eye. The remainder of the examination was essentially negative.

Treatment consisted of removal of the glass foreign body with a needle under topical tetracaine (Pontocaine) anesthesia. This was followed by swabbing the bulbar and palpebral conjunctiva with a moist cotton swab in an effort to remove any unseen glass particles. Chlortetracycline (Aureomycin) ointment was instilled and a patch applied.

The patient was seen every few days, and on June 19 the epithelium finally was intact when examined by slit lamp with fluorescein. The conjunctiva was still inflamed, and cortisone ointment was prescribed every three hours. On June 21 pain recurred. On examination, the epithelium was observed to have broken down centrally. Two days later this area was healed; but from 5 to 7 o'clock a marked increase in visibility of corneal nerves was noted, and the overlying area stained heavily with fluorescein. Chlortetracycline and cortisone ointment and a patch were applied. On June 24 the cornea had innumerable areas denuded of epithelium. The corneal nerves were swollen and white throughout the lower two-thirds of the cornea. The stroma was hazy. Visual acuity was finger counting at 3 ft. (90 cm.). There was never any flare. Finger tension was always normal. Gifford's iodine solution was applied and the corneal epithelium carefully scraped off. The eye again was patched. The epithelium continued to break down. Therefore, on June 30 the treatment was repeated, and, in addition, the cornea was completely covered with a conjunctival flap. Both eyes were closed for five days, at which time the epithelium was intact. A stromal haze persisted. On July 8 the dressing was eliminated. Treatment with instillation of 2 drops of 20% cortisone solution every two hours when awake and of cortisone ointment at bedtime was ordered. Visual acuity at this time was finger counting at 5 ft. (1.5 meters).

From the Department of Ophthalmology, Ponce de Leon Eye and Ear Infirmary.

The patient was asymptomatic until July 22, 1952, when pain recurred and a minute central area stained. This area increased to about 4 mm. in diameter by July 26, 1952, and then decreased and was healed by Aug. 1, 1952.

Vascular infiltration of the corneal stroma was most marked on July 31, 1952, and then receded by Aug. 8, 1952. The cortisone regimen described above was uninterrupted through this period. The corneal haze continued to diminish; the injection disappeared, and the epithelium remained intact. Visual acuity on Dec. 20, 1952, was 20/200, not improvable with lens or a pinhole glass. The upper third of the cornea was completely clear, and a slight haze persisted in the superficial stroma of the lower two-thirds.

The draft board and the compensation aspect are probably more to be blamed for the visual acuity than the residual slight corneal haze.

The swelling of the corneal nerves was striking; but it is not rare in inflammatory corneal processes, and hence should not be construed as a specific effect of beryllium.

Cortisone given topically had no observable effect on healing or on preventing the epithelial erosions from recurring.

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Annual Reviews

THE LIDS, LACRIMAL APPARATUS, AND CONJUNCTIVA

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IOWA CITY

THIS REVIEW of the literature is intended to cover as many of the papers published from the summer of 1952 to the summer of 1953 as is possible. The large volume of material has been covered as completely as possible, with aid in translation by Drs. Hermann M. Burian and Frederick C. Blodi. The papers on "Woody Conjunctivitis" and "Ocular Accidents of Vaccinal Origin" were translated by Dr. Bernard Schwartz.

The outstanding contribution to the literature on Lids and Lacrimal Apparatus is the fifth volume of the "Text-Book of Ophthalmology," written by Sir Duke-Elder. Since this beautiful, complete volume was published in 1952, I will use it as the final authority.

THE LIDS

Congenital Anomalies

Anomalies of the Palpebral Fissure.—Edeskuty [1] suggests that the orbicularis is responsible for the abnormal positions of the lids, particularly the lower lids. He thinks that the muscle fibers move toward the lid margin and turn the tarsus inward. He also discusses the development of senile entropion and ectropion and separates the orbicularis into areas in order to explain the development of various positions of the lids.

Duke-Elder [2] (p. 4365) states that the abnormal positions of the lids and palpebral aperture are "generally associated with mal-development of the external canthal ligament, or it may be a part of a general failure in the development of the face."

Bischler [3] reports an interesting observation in a girl, 4 years old, with a pseudo-Graefe-phenomenon. She describes the case as a congenital anatomical fixation of the upper lid. Her case is the fourth reported. The lid was slightly closed and did not close in sleep.

Adda, in 1938, cited by Bischler,[3] thought it was a retraction syndrome.

Congenital Ptosis (Blepharoptosis).—Blepharoptosis [2] (pp. 4677 and 5134) is a deformity with a strong hereditary tendency and may be of seven types:

1. Simple ptosis
   (*a*) Affecting the lid only
   (*b*) Combined with paresis of the homolateral superior rectus
2. Ptosis associated with other deformities of the lids, i. e., epicanthus and blepharophimosis
3. Ptosis complicated by varying degrees of external ophthalmoplegia
4. Sympathetic ptosis
5. Synkinetic ptosis, i. e., jaw-winking
6. Periodic ptosis, i. e., cyclic oculomotor spasm
7. Intermittent pseudoptosis, i. e., retraction syndrome

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Posner [4] thinks that ptosis has a dominant heredity and is transmitted by either sex. There is little doubt that ptosis is hereditary and readily transmitted [2] (p. 4679).

Berke [5] in the past years has discussed the causes of ptosis; however, during the past year he has reported his extensive experience with operative procedures. He states that there are almost 90 different operations for the correction of ptosis. He divides the procedure into three groups:

1. Frontalis muscle type of ptosis operation
2. Superior rectus muscle type of ptosis operation
3. Shortening of the levator palpebrae muscle

He states that the most important consideration is to determine the nature of the type of ptosis. He divides his types of ptosis into four groups, while Spaeth, cited by Berke, divides congenital ptosis into nine groups. He wishes to emphasize that "the nature of ptosis in a given case determines the nature of the result." All cases of congenital ptosis may be classified into one of four groups:

1. Ptosis with normal superior rectus muscle function
2. Ptosis with weakness of the homolateral superior rectus muscle
3. Ptosis with the jaw-winking phenomenon of Marcus Gunn
4. Ptosis associated with blepharophimosis

In Group 1 resection of the levator produced good results in from 57 to 78% of the cases, depending on the levator action present. In Group 2 these figures drop to 30 to 50%, respectively. In Group 3 resection of the levator is contraindicated. Here, the jaw-winking must be destroyed by excising a piece of the levator and the ptosis corrected by other means. Group 4 presents a surgical challenge for the correction of the blepharophimosis, as well as the ptosis.

Berke [6] developed a new clamp for holding the levator muscle during a resection of the muscle. The clamp is used in a simplified operation similar to that described by Blaskovics 30 years ago. He [7] reviews the pitfalls of the Blaskovics operation and presents the anatomical picture of the lid and the structures adjacent to the levator palpebrae. A study of 91 cases indicated that a preoperative examination should be complete and detailed. The amount of ptosis in the acquired cases varied from 3 to 8 mm., with an average of 4.8 mm., while in the congenital cases it measured from 2 to 8 mm., the average being 3.8 mm. In 57 of the 78 eyes the superior rectus was normal, and in 21 it was weak. The amount of levator action may be determined by the superior rectus action. The operative technique is fully described in the paper, but a short description will be offered.

The upper lid is retracted upward, and the globe is pulled down. An absorbable surgical suture is placed under the superior rectus muscle. The upper lid is doubly everted and the conjunctiva undermined from the lateral side. The conjunctiva is then freed to the edge of the tarsus and cut free from the tarsus with scissors. Sutures are placed in the free edge of the conjunctiva. Müller's muscle and aponeurosis now are undermined from the septum orbitale and the levator clamp applied. The tendon and Müller's muscle are severed from the tarsus. The levator tendon is then isolated so it will come out about 10 to 12 mm. The conjunctival sutures are then passed through the levator and tied. The upper edge of the tarsus now passes through the sutures and out through the skin, where the sutures are tied

over a rubber at the desired fold. A Frost suture is then placed. Berke [7] describes some of the complications occurring during the operation—the superior rectus is the most vulnerable. The final results are given in 72 patients followed from three months to five and one-half years. Correction of acquired ptosis averaged 4.3 mm., with a resection averaging 20 mm. In congenital ptosis the average correction in 78 eyes was 2.3 mm., with an average resection of 18 mm. The postoperative complications are (1) temporary overcorrection, (2) temporary superficial keratitis, (3) extraocular muscle anomalies, and (4) lagophthalmos in looking down.

Scott [8] presents the results of a study of 25 cases. He agrees with Berke as to the types of procedures. He says operations with the frontalis are the most popular and the most frequently described. He thinks that the disadvantages of the operations using the superior rectus make them undesirable. Lagophthalmos with exposure keratitis, especially during sleep, may be troublesome. The upper lid is unduly arched, and there is frequent postoperative vertical muscle imbalance. He believes that the best operation is resection and advancement of the levator. He states that the operation should be done when the child is 3 years of age. If the ptosis is of a severe type, it should be done earlier. He did 25 operations, 20 with perfect or near-perfect results; 3 with undercorrection, and two were complicated but no result was obtained.

Sarwar [9] describes a new operation and reviews many of the other types of operations.

Ptosis operations

1. Removal of skin only
2. Use of the frontalis to lift the lid
   (*a*) Anchoring the lid to the frontalis by a strip of skin
   (*b*) Anchoring the lid to the frontalis by a suture (Hess and others)
   (*c*) Anchoring the lid to the frontalis by a fascia lata suture
   (*d*) Attaching the levator palpebrae to the frontalis
   (*e*) Frontalis strips brought down and anchored to the lid (Fergus, modified by Roberts)
3. Surgery on the levator tendon (Everbusch, Blaskovics, and modifications)
4. Use of the superior rectus as an elevator (Motais, Graeves)
   (*a*) Directly by attaching the muscle to the tarsus (Kirby, Weiner, and others)
   (*b*) Lid sutured to the globe (Young)
   (*c*) Fascia lata sling (Dickey)
   (*d*) Strip of tarsus passing behind the rectus (Trainer)
   (*e*) Strip of orbicularis attached to the superior rectus (Wheeler)
5. Strips of orbicularis from near the lid margin slung up to the frontalis (Rees, Hunt-Tansley)

Sarwar's new technique uses a strip of frontalis which he passes down through a tunnel under the skin to the upper margins of the tarsus; it is then brought back to the point of origin in the frontalis. The strip is tied down with sutures with the lid in position. A Frost suture is then placed in the lower lid. He reports that 8 of 10 patients had good results. I think that this operation should produce a notch in the lid at the point of attachment of the tarsus.

Kreibig [10] believes that the Friedenwald-Guyton sling suture is the best, since it is easily performed, does not lead to scars, gives good results, and can be repeated.

None of the operations are wholly satisfactory in my hands. I think the resection of the levator in suitable cases is the operation of choice. The best results are obtained in the bilateral cases and the poorest in the cases of jaw-winking with weakness of the superior rectus.

An interesting case of functional post-traumatic ptosis was recorded by Schachter.[11] The functional element was established by clinical analysis and improved by physical therapy. We as physicians should always keep this side in mind in any case of acquired ptosis.

Entropion

Entropion may be listed as acquired, congenital, cicatricial, mechanical, organic, senile, spastic, and voluntary [2] (pp. 4661 and 5182). Since the cause of congenital entropion is discussed above, this portion will deal primarily with the extensive literature on surgery for the relief of entropion. Fox [12] has modified the technique of his operation for senile entropion originally reported in October of 1951. The operation is described in some detail, in which a triangular piece of tarsus and conjunctiva is removed with the apex toward the lid margin. A vertical elliptical piece of skin and orbicularis muscle is removed temporal to the external canthus. After the skin and muscle is separated from the tarsus, the lips of the wound are easily closed.

Lebensohn [13] has a modified Machek's operation in which he utilizes a skin sling to support the lower lid. Machek's operation was primarily intended for the correction of congenital ptosis. Lebensohn ingeniously has applied a similar procedure to repair entropion. Parallel incisions are made in the skin at the lid margin and 4 mm. below it. The skin is left intact at the central lid margin for a distance of 5 mm. The two pieces of skin are lifted and denuded by trichloroacetic acid. Double-armed silk sutures are placed at each end of the skin; these ends are threaded through tunnels reaching below the rim of the orbit. Lebensohn cautions against overcorrection. He has done the operation in 12 cases, with good results. While these operations are primarily intended for spastic entropion, Pavišić [14] has described a procedure for cicatricial entropion which is done from the conjunctival side. The tarsus is cut through 2 mm. from the lid border and freed from the conjunctiva. Three mattress sutures are placed on the conjunctival side through the lid border, thus everting outward the cilia and the lid border. The defect is filled with a 2 mm. strip of mucous membrane from the lip, which is sutured between the lash line and the border of the tarsus. He claims good results for the severest types of cicatricial entropion and trichiasis.

Van Manen [15] describes a combination instrument, which he calls the North African eyelid plate, designed to help perform operations on the tarsus. The plate is intended to hold the eyelid everted and to control bleeding while performing an operation similar to Ewing's. Since most of the cicatricial entropia are produced by trachoma in many areas of the world, Pavišić [16] has described two ingenious methods of correcting entropion and coloboma produced by tarsectomy. In one, he takes a piece of ear cartilage and uses skin from the lid to cover the cartilage graft. In the other, he [17] uses mucous membrane from the lip similarly to his previously described method with slight modifications.

Boase [18] performs tarsectomies for the relief of cicatricial entropion. He operates on about 2,000 lids a year. The operation is essentially a modification of Saunders' or Heisrath's operation. He performs a subtotal excision of tarsus with its adherent conjunctiva. He states that he does 20 bilateral tarsectomies in a morning. His indications for performing tarsectomies are (1) bad cases of Grade II trachoma, (2) vernal conjunctivitis with large vegetations, (3) spastic entropion in children (induced by malarial headaches), and (4) moderate degrees of ptosis. He also does tarsectomies for entropion of the lower lid; however, this operation is more difficult to perform and is rarely necessary. He also uses Cruickshank's entropion forceps. He stresses that the incision on the conjunctiva should be made in the subtarsal sulcus from canthus to canthus and not near the lid border. No lash follicles should be seen. After the tarsus is removed, it is closed with three mattress sutures, the upper border of the tarsus being brought down to the lid border. Although I am sure tarsectomy is one of the commonest operations, it is not without danger of severe keratitis.

Hartmann [19] still likes to use electrocoagulation, which he described 10 years ago. Now in the severe cases, he uses a modification of Wessely's operation. He prepares the bridge of skin by making a Y-shaped incision in the lower lid. The skin is used to pull the lower lid downward. While many surgeons remove portions of skin, tarsus, and other substances, Foster [20] wishes to preserve these structures.

Ectropion

Duke-Elder [2] (p. 5188) classifies ectropion into five types. They are spastic, senile, paralytic, mechanical, and cicatricial. The congenital type has already been considered.

Foster [20] states that in the repair of the eyelids, either from congenital or acquired lesions, use of sliding skin flaps, tarsectomies, and other operations are not suitable. He says the loss of the tarsus gives an immobile, shapeless lid, which is more subject to either entropion or ectropion. He prefers a modification of the Büdinger-Müller lid plastic operation, which had been described in 1902. Büdinger replaced the outer third of the lower lid with a temporal pedicle flap, in which he sewed a curved piece of cartilage taken from the scaphoid fossa. Müller replaced all the layers at once from a piece of ear cartilage. Foster showed 12 completed cases. Bishop and Schrire [21] used a tubular pedicle graft for a similar post-traumatic cicatrical ectropion repair.

Since many tubular grafts are described for the reconstruction of eyelids, Conway, Stark, and Kavanaugh [22] present comments on the development of the temporal flap. These flaps are frequently used for the repair of both the upper and the lower lid, the lip, the cheek, and the nose. They showed by angiography of the skin through the common carotid artery of the cadaver that the superficial temporal artery contributes mostly to the scalp, while the temporal flap should contain either the frontal or the parietal superficial branches. They stress the continued refinement of pedicle flap technique. Pedicle grafts are also reported on by Gantchev,[23] using Filatov's method.

In a symposium in "Ocular Injuries," a number of references are made to reconstruction of the lid. Leahey [24] stresses that the ophthalmologist should not fail to treat the shock which is present following burns of the face and eyelids. He says

that burns should be covered by sterile dressings and that blebs should not be broken. He feels that boric acid ointments should be avoided because of the toxicity. He feels that pressure dressings by means of a stockinette over the head with openings for eyes, nose, and mouth are indicated. Since most burns result in ectropion, it is included in this section. The dressings are left on as long as they are dry, clean and the body temperature remains normal. They are not removed for from 7 to 10 days. If infection is present, the dressings should be changed daily. As sloughs develop, they are allowed to separate spontaneously, and they may be grafted in three weeks. He states that split-thickness grafts take well on a granulating base, but that the appearance is inferior to the full-thickness graft. He applies immediately a full-thickness graft if the burn is clean and not too large. The burn should be deep and the full-thickness graft sutured to normal skin. He states that sliding grafts may also be used. Spaeth [25] discusses the late repair of ectropion and entropion and the contracted socket. He thinks that most surgery should be done at one time, and not divided into stages. Enucleation of the eye may have to be done in order to help with the repair. Scheie [26] says:

> Repair of lacerations of the lids should be done during the first few days after an injury, because defects which are easily repaired with no deformity at the time of injury or shortly thereafter become extremely difficult to correct later and are associated with severe deformity and incapacity.

The "halving" operation of Wheeler is suggested by Scheie to correct lid lacerations and prevent notching. Scheie describes an ingenious method for repairing the tears of the medial canthus when they involve the lower canaliculus by using nylon sutures tied in a loop. Hogan [27] briefly discusses injuries to the eyelids, stating that ecchymoses are common but rarely serious. They may become bacterially contaminated, and emphysema may also develop.

McPherson [28] objects to the methods of both Reese and Hughes in reconstruction of the lower lid. By combining the two methods, McPherson thinks he has a better procedure. He uses the tarsus from the upper lid as a tongue placed into a defect in the lower lid, the skin over the tarsus being supplied by a lateral sliding graft. In his one case, a satisfactory result was obtained.

Shukla [29] used skin from the upper lid to re-form the lower fornix of a deformed socket.

One of the many interesting subjects I read was Langworthy's [30] discussion on blepharospasm and facial tic. He well documented four cases with severe blepharospasm. Like the heroine in Hawthorne's story [31] who wore the letter "A" for adultery, these people display a symbol for others to see. They are not able to open their eyes because they cannot look at the facts. They form a separate group of conversion hysterias. All four patients were perfectionistic persons, concerned with the problem of giving and receiving; each had a feeling of never receiving a fair share of the bargain. They appeared to try to get the better of the bargain by methods not completely honest. The blepharospasm appeared rather suddenly, after the patients had been rejected by some person close to them, and money was important in all discussions.

Silato's [32] cases of unilateral spasm of the upper lid were considered to be produced by spasm of Müller's muscle. Graves's disease was considered the cause.

Inflammation of the Lid Margins

Blepharitis.—Infectious dermatoses are classified by Thygeson [33] as follows:

1. The pyodermas
   (*a*) Marginal blepharitis
   (*b*) Impetigo contagiosa
   (*c*) Ecthyma
   (*d*) Sycosis vulgaris
   (*e*) Infectious eczematoid dermatitis
   (*f*) Erysipelas
   (*g*) Diphtheria
2. Diseases of the sebaceous glands
   (*a*) Meibomitis
   (*b*) Milium
   (*c*) Seborrheic dermatitis
   (*d*) Rosacea
3. The eczema group of dermatoses
   (*a*) Atopic eczema
   (*b*) Contact dermatitis
4. Dermatoses due to photosensitivity
   (*a*) Xeroderma pigmentosum
   (*b*) Hydroa vacciniforme
   (*c*) Acute solar dermatitis, or sunburn
5. The dermatomycoses
   (*a*) Ringworm of the scalp
   (*b*) Trichophytosis of the scalp
   (*c*) Favus
   (*d*) The epidermophytoses
   (*e*) The dermatophytids
   (*f*) Torulosis
   (*g*) Blastomycosis
   (*h*) Sporotrichosis
   (*i*) Actinomycosis
   (*j*) Cutaneous moniliasis

Thygeson [33] writes that staphylococci and streptococci are associated with most of these infections. He states that marginal blepharitis, both squamous and ulcerative, is the commonest. It is associated with hordeolum and chalazion. Impetigo contagiosa, ecthyma, sycosis vulgaris, and infectious eczematoid dermatitis may be associated with blepharitis. No discussion of blepharitis would be complete without referring to the chapter in Duke-Elder [2] (p. 4949).

Parker,[34] Ainslie,[35] and Azzolini [36] discuss their treatment of blepharitis, the agents varying from antibiotics to x-rays. DeVeer [37] describes the pathology of a large chalazion. Borges Dias [38] states that ocular tissues, particularly with skin, may be sensitized locally or be involved in a general process of sensitization to any foreign substance.

Vidal and Weil [39] present an interesting discussion of seborrheic blepharitis. They claim that a lipid secretion combined with the infection produces the disorder. The oily secretion arises in the Meibomian glands and is largely oleic acid. In lipid blepharitis the glands secrete a paraffin-like stearic acid compound, which tends to obstruct the necks of the glands. The authors believe the disease is due to a faulty lipid metabolism with the development of allergic hypersensitivity.

Von Fuchs [40] thinks the orbital abscess and the lid abscess must be differentiated.

Fazakas [41] is interested in the fungi, which may be either primary or secondary, associated with infectious blepharitis. He has found the Meibomian glands infected in three patients, one with Acrostalgamus cinnabarinus Corda, one with Sterygmatocystis nigra, and one with Acrothecium hominis Oláh. The lid margins he found infected with Achorion Schönleini (favus), Microsporon felineum (cats), and Trichophyton. Mendelblatt [42] reviewed the literature on Candida albicans and found that it produces eczematoid monilids, vesicles, and pustules on the eyelids.

Mitsui (personal communication) reports that C. albicans was capable of sensitizing rabbit eyes after repeated instillations. The work was done by Hanabusa.[43]

Swinny [44] states that the periorbital region is the frequent site of contact dermatitis. He says that history of the contactant is the most important, whereas contact testing may not help. Of 63 patients with contact dermatitis of the lids, 85% were females and only 15% were males. Cosmetics play a major role, while cream-based shampoos are also important. Eighty-eight per cent of the patients were between 20 and 60 years of age.

Fuchs [45] described a peculiar scar of the upper lid on many people in China. The scars are 2 to 3 cm. long and 0.3 to 0.5 mm. broad, usually above the skin fold. He discusses several possible causes and discards all of them.

Anderson and Asboe-Hansen [46] present a case of severe elephantiasis of the face following eczema of the scalp and face. The lids were thick, firm, pink, and pasty to palpation. Peripheral blood showed an increase in eosinophiles. A biopsy showed collagen fibrils with a ground substance of mucopolysaccharide, consisting chiefly of hyaluronic acid. Injection of hyaluronidase into the lid did not reduce the swelling. Corticotropin was given intramuscularly, 10 mg. four times a day for eight days, softening the tissue but not removing the swelling. The swelling had to be removed surgically.

Von Nordheim and van Baak [47] found that injection of 5 V. R. U. of hyaluronidase daily for three weeks into the upper lid and brow gave a good response. The edema had been present for 10 years. Since the specific cause in these cases was not proved, it seems impossible to draw any conclusions. The condition of severe prolonged edema of the lids and brow should be produced by blockage of the lymphatic circulation by chronic inflammation. For an excellent discussion on edema of the lids, see Duke-Elder [2] on this subject (p. 4784) and on elephantiasis (p. 4993).

Oculocutaneous Lesions.—The volume of literature on this confusing subject is divided into two parts: the diseases occurring primarily on the lid, i. e., Stevens-Johnson disease, and those occurring primarily on the conjunctiva, i. e., pemphigus. The latter will be discussed under "Conjunctiva."

Bluefarb [48] discusses the entire group of involvements of the skin and the lids, including the common forms of blepharitis, contact dermatitis, and many others, in an all-inclusive review.

Orme [49] describes a case of a 5-year-old girl who died with what he called erythema multiforme exudativum after one month of hospitalization. She had had frequent small styes, which had been treated with penicillin. An acute, swollen, red knee joint developed nearly every year prior to her admission. Her last hospitalization began with an acute upper respiratory infection and a temperature of 104 F. The fever dropped on penicillin therapy, but she developed a sore throat and a grayish membranous lesion in the mouth. Penicillin was stopped and she was given chlortetracycline (Aureomycin), and her eyelids became severely involved. She developed bullous lesions on her skin and hands. Corticotropin was given and the eyes improved; however, her temperature rose to 105 F., and she died in shock. She may have had a perforated gastric ulcer. From the description, I believe that she had acute pemphigus.

Agostos, Reeves, Shanks, and Sydenstrecker [50] discuss the Stevens-Johnson syndrome from the viewpoint of etiology, and they feel that the theory of an allergic etiology is the most tempting. They feel that drugs or bacterial products may sensitize the patient. Sulfonamides and penicillin are most incriminated. They

report two cases treated with corticotropin with good results. Pritchett and Austin [51] report a disease they called Stevens-Johnson disease in a mild form, which began in one twin and developed later in a second twin. Chlortetracycline (Aureomycin) was used in one case, but oxytetracycline (Terramycin) did not prevent the development of the cutaneous eruption, vesicular stomatitis, or conjunctiva-lid involvement in the second twin. However, they felt that oxytetracycline decreased the length of the illness.

Neller and Lang [52] think that mucocutaneous ocular syndrome is the best name for the large group of diseases including Stevens-Johnson, Behcet's, Reiter's, and related diseases, and that they are all variants of erythema multiforme. They report that chlortetracycline was beneficial within 48 hours. On the other hand, Mecklin and Saunder [53] report startling results with cortisone. Fuiducci, Frank, and Franks [54] feel that chlortetracycline and oxytetracycline is the best form of therapy. Numerous authors add to the confusion concerning Stevens-Johnson disease (Venturi,[55] Hauge,[56] Pereyra,[57] and Contino [58]). For a discussion of the pathology, see "Conjunctiva").

Virus Diseases of the Lids.—Since viruses may involve several portions of the eye and they are well covered in a table from Cavarra and Bietti's [59] monograph, part of the table is reproduced here.

Vaccinia of the Lids.—Sédan, Aurgaud, and Guillot [60] had an opportunity to study several cases of accidental vaccinia infections. Of 850,000 people vaccinated, they found 19 with accidental ocular involvement. Although Morax thought that the virus of vaccinia was not present in these lid lesions, numerous authors have isolated the virus from the ocular lesions. They feel that the complications usually appear in six to eight days. Of the 19 cases, 9 were of the lids; 6, corneal; 3, oculomotor, and 1, of iridocyclitis. Accidental infection may occur at any time, even after a number of revaccinations. They claim that chlortetracycline and cortisone are effective therapy.

Frampton and Smith [61] describe a typical vaccinia in a 14-year-old girl who had not previously been vaccinated. They recovered the virus from the lid lesions. Chloramphenicol (Chloromycetin) was effective.

Kimura, Thygeson, and Geller,[62] in a series of cases of experimental vaccinia keratoconjunctivitis, demonstrated that cortisone given prior to the infection may increase the severity of the infection, especially increasing the dissemination of the virus.

Givner [63] wishes to emphasize that finger-to-eye contamination may occur. I have stressed that the vaccination should be placed well back on the arm, where it is difficult to reach. In my experience, most accidental vaccinia infections have occurred from pustules on other persons or when the patient is inoculated on the leg.

Herpes Zoster Ophthalmicus.—Herpes zoster ophthalmicus is caused by a specific virus related to varicella and distinct from herpes simplex, which attacks the Gasserian ganglion. The frontal branch of the ophthalmic nerve is usually involved [2] (p. 4886). The post-herpes-zoster pain is little understood.

Dolan and Bucy [64] report on a patient who had the entire sensory root of the fifth nerve divided central to the ganglion. He had total anesthesia of that side of his face. However, he developed herpes zoster ophthalmicus after trauma. The findings were classic but without pain. The skin showed areas of slough but still

no pain. They report that Gardner had a similar case. They point out that the pain is mediated by the trigeminal nerve and must have a central connection. Tatlow [65] presents a comprehensive review of 58 cases which he studied in detail. Several forms of treatment are discussed, including chlortetracycline, oxytetracycline, and chloramphenicol, but I have not been impressed with this treatment. Micheils [66] and

*Tabulated Summary of the Principal Ocular Localizations in the Disease for Virus and Rickettsia Pathogenic for Man**

	Lids (Skin)	Lacrimal Glands	Canaliculus; Lacrimal Sac	Conjunctiva	Cornea
Herpes simplex or febrilis	++	....	....	++	++++
Herpes zoster	+++	....	....	+++	++
Varicella, or chickenpox	+	....	....	++	++
Smallpox	+++	....	....	+++	++
Para-smallpox	....	....	....	....	....
Cowpox	++	....	....	++	++
Trachoma	+++ tarsus	++	++	++++	+++
Conjunctivitis from inclusion body	....	....	....	++++	+
Lymphogranuloma venereum	++	....	....	+++	+++
Psittacosis and virus pneumonia	....	....	....	+	....
Benign lymphoreticulosis	++	....	....	+++	....
Measles (morbilli)	+++	+	+	+++	++
German measles (rubella)	++	....	....	++	+
Epidemic keratoconjunctivitis	....	....	....	++++	+++
Epidemic encephalitis	....	....	....	+	+
Lymphocytic choriomeningitis	+	....	....	+	....
Rabies	....	+	....	++	+
Influenza	+	+	+	+++	++
Common cold	....	....	....	+	....
Epidemic parotitis (mumps)	....	++	+	+	+
Yellow fever	++	....	....	++	....
Dengue	++	....	....	+++	+
Sandfly fever	....	....	....	+++	....
Rift Valley fever	....	....	....	+	....
Colorado tick fever	....	....	....	+	....
Infectious mononucleosis	++	....	....	++	....
Infectious hepatitis	....	....	....	+	....
Molluscum contagiosum	++++	....	....	+++	++
Verruca	++++	....	....	+++	++
Newcastle's disease	....	....	....	++++	....
Foot and mouth disease	+	....	....	++	++
Exanthematic typhus and murine typhus	....	....	....	+++	+
Rocky Mountain fever	+	....	....	+	....
Mediterranean exanthematic fever	+	....	....	++++	....
Rickettsial pox	++	....	....	....	....
Tsutsugamushi fever	++	....	....	+++	....
Recurrent fever	....	....	....	+	....
Pemphigus	++	....	....	++	++
Reiter's syndrome	....	....	....	++++	+
Behcet's syndrome	....	....	....	+	+
Stevens-Johnson syndrome	....	....	....	+++	++
Benign lymphogranulomatosis (Besnier-Boeck-Schaumann disease and Heerfordt's syndrome)	+	++	....	++	+
Malignant lymphogranuloma (Hodgkin's disease)	+	++	....	++	+
Leukemia	++	+	....	++	+

* From Cavarra and Bietti.[59]

many others, reporting prior to 1952, are impressed with chlortetracycline in the treatment of zona. Cavarra's [59] chapter on herpes zoster (p. 217) also discusses cobra venom and alcohol injection.

Since there appears to be a close relationship between herpes zoster and varicella (chickenpox), I was interested in Glotzer's [67] case of herpes zoster ophthalmia occurring simultaneously with varicella in a 66-year-old man. No history of contact with either disease could be obtained.

Strong [68] followed a case of zona in a 48-year-old lieutenant who had severe zona ophthalmica with a corneal ulcer in May of 1947. He states that the patient had no treatment, but in 1949 developed Parkinson's syndrome and died in September of 1950. Strong reviewed the literature and found that among the complications of herpes zoster, motor nerve paresis, myelitis, meningitis, and encephalitis occur. Disseminated sclerosis and right hemiplegia are also discussed. I have also seen severe complications from herpes zoster ophthalmicus, and mild meningitis or encephalitis is common.

Zelaski [69] showed an 18-month-old Negro child with a large tumor of the upper lid which resembled a chalazion. It proved to be molluscum contagiosum. No mention was made of conjunctival or corneal involvement. Duke-Elder's [2] discussion is complete and concise (p. 4892).

Tumors of the Eyelids

One of the most interesting pathological studies on xanthelasma palpebrarum is presented by Wolff,[70] who thinks it is produced by a tumor of the sebaceous glands. He points out that, while this is not a new concept, he believes that the demonstration of single sebaceous cells occurring in the epidermis is new. He states that under certain conditions these cells multiply and produce the yellow masses called xanthelasma. The nuclei of these cells undergo the same changes as in an ordinary sebaceous gland. Since the hair follicles are few, the cells do not develop into glands but form sheets. They form in the region of the inner side of the eyelids because of the special character of the skin. Duke-Elder [2] has accepted this theory (p. 4990).

Sebaceous cysts are also common in the region of the inner canthus, as reported by Grönvall.[71] He collected 375 cases, in which the cysts occurred most frequently in elderly people. They occurred near the medial canthus and the root of the nose, following the cleft lines of the skin of the face.

From my own experience and that of Ginsberg,[72] Fontana,[73] Contino,[74] Wakeley,[75] and Hunt,[76] epithelioma also occurs commonly at the inner canthus. The tumors are sometimes extremely malignant and spread rapidly, as in the case reported by Fontana.[73] Likewise, they may metastasize widely, as in the case of Hunt.[76] Hunt's [76] case is interesting in that the entire lower lid was involved, from inner to outer canthus. After considerable surgery, the patient died, and autopsy revealed metastases to the lungs, dura mater, sphenoid bone, femur, clavicles, and thoracic and lumbar vertebrae. Every bone examined at autopsy contained tumor. Hunt [76] suggests that the metastases may have occurred because of the unavoidable bleeding at the time of operation. Wakeley [75] and Ginsberg [72] use both surgery and irradiation in treatment of epitheliomas; Contino [74] prefers surgery.

Several rare tumors are reported. Provasi [77] saw three cases of syringioma of the eyelids, which arose in the sweat glands. Stankovitch [78] reports on a woman aged 60 with tumors on all four lids. The preauricular and submaxillary glands became

enormous. Death was rapid, and biopsy showed a round-cell sarcoma. Brand and Kadas [79] report a myeloid tumor of the eyelids. Two interesting metastatic tumors of the lid are presented.

Vrabec [80] demonstrated retrolymphatic metastases of a primary tumor of a parotid into the lid, where it involved the deep tarsus.

Cowan [81] describes an interesting case of metastasis of an adenocarcinoma of the stomach to the lid. The tumor of the lid had been removed twice as a chalazion, and with the recurrence a biopsy of the lid was done, the findings suggesting an adenocarcinoma of the stomach. The patient had an adenocarcinoma of the stomach with metastases to the liver, as well as to the eyelid.

Rudowski and Morawiecki [82] describe an ingenious method of treating cavernous hemangiomata of the eyelids. They describe six cases in which the lesions were injected with boiling water and the results were gratifying. They warn that the water should be injected deep enough to prevent necrosis of the skin.

LACRIMAL APPARATUS

Embryology

Cassady [83] publishes the pictures from an exhibit which he had presented and adds to this material some new observations. He states that the lacrimal passages arise from an epithelial cord imbedded in the mesenchyme. This cord is first apparent in the 7-mm. embryo as a thickening in the ectoderm at the naso-optic fissure. The ectoderm grows and extends to reach the ocular and nasal extremities. He states that 73.3% of the lumina are not open at birth. Cowan [84] agrees that a high percentage of lumina are closed at birth. He thinks that probing is frequently necessary. I usually wait until the baby is 3 or 4 months old or infection intervenes.

Congenital Anomalies

Sjögren [85] discusses congenital alacrimia, stating that Morton, in 1884, had reported the first case of congenital bilateral absence of lacrimation. Since then, 14 cases have been reported from the ophthalmic literature. Several have been diagnosed as absence of the lacrimal gland and hypoplasia of the gland, and some were bilateral, while others were unilateral. He states that neurogenic disturbances were frequent. Commonest were palsies of the extraocular muscles.

Coverdale [86] reported at the same meeting in Australia and discussed a patient he had reported on in 1948, a woman aged 36, who had never had tears and began to show corneal changes at the age of 26.* Since I had an opportunity to see a number of the cases reported by Riley,[87] I feel it is worth while to describe a condition which may be similar to those described by others, which Riley has called familial autonomic dystrophy. To date, Riley has collected 33 well-documented cases. All patients were of Jewish extraction, and most of the following symptoms are present: defective lacrimation, excessive perspiration, abnormal drooling, emo-

* He presented an interesting family—the mother, aged 33, a 1-year-old baby, and a 26-year-old brother. The baby had no tears; the mother had very low tear function, with rheumatism of the wrist, dry mouth, and chronic indigestion. The brother had a dry mouth and many mucous shreds in the eyes, but a reasonably good tear function. Since lack of tears in babies is variable and there is no definite time for the development of tears, Sjögren [85] warned against the diagnosis of congenital alacrimia. He recalled a case, reported by Sommers in 1903, of a 2½-year-old child without tears who developed tears at about the age of 3 years.

tional instability, general motor incoordination without specific neurological findings, and relative indifference to pain. Blotching of the skin of the head and shoulders is common. Corneal ulceration probably is secondary to defective lacrimation, and corneal hypesthesia has been present in about one-third of the patients. I have performed lid adhesions in four patients with beginning corneal changes, with gratifying results. Riley further states that mental retardation is a problem in more than one-half of the patients. Convulsions presented such a prominent symptom that exploration was done in five patients for the possibility of pheochromocytoma, but none was found. Of the 33 patients, 10 have died of aspiration pneumonia, and some with intermittent hypertension. Riley thinks that there is no change in the condition with the passage of time and that it is undoubtedly familial.

Congenital fistulas and diverticula of the canaliculi were reported by Penzani [88] and Kubik.[89]

Tears

DeRoetth [90] feels that tears are a secretion of the lacrimal gland without the admixture of secretion of the other glands of the conjunctiva. The substance called tears is a watery fluid which is present in the conjunctival sac and is a production of the lacrimal gland, the accessory lacrimal gland, the Meibomian glands, and the mucous glands of the conjunctiva. The Schirmer method for testing tears is rather crude, but is the only practical one. DeRoetth examined 827 persons and determined that the lacrimal secretion decreases with age and begins to fall off between 20 to 50 years of age. He concludes that the amount of lacrimal secretion is normal for that particular eye if the eye is functioning without discomfort.

Pedersen-Bjergaard and Smidt [91] present data concerning the normal pH, vapor pressure, and conductivity of tears. They collect tears by pulling down the lower lid and collecting them with a capillary pipette. If no tears are present, the eye is exposed to formalin. They examined 25 females and 25 males, varying in age from 9 to 51 years. The conductivity test determinations were equivalent to 0.90% NaCl. The vapor pressure tests were equivalent to 0.93% NaCl. The pH was an equivalent of 7.4.

Balik [92] collected data on 32 patients with keratitis sicca and compared them with the average results for 30 so-called normal patients. He claims that Bang's paper should be used to determine tear function. The increase in weight of the paper strip before and after being placed in the conjunctival sac designated the amount of tears. Although Balik carried out his weights to the fourth decimal place, he determined that the increase in weight for the normal was from 77 to 61 mg., while in keratitis sicca the increase in weight was from 30 to 40 mg. He states that the chief hydrolyzed protein in tears is mucin and that there is no difference in the NaCl content between normal and abnormal tears. He thinks that as the tear function decreases the amount of protein increases. DeRoetth [93] accepts the hypothesis of the lacrimal gland as having a role in the pathogenesis of keratoconjunctivitis sicca and keratitis filiformis. He states that many patients complain of dry eyes and have low tear function, as demonstrated by the Schirmer test. Thirty-one of these patients did not have keratoconjunctivitis sicca but had burning and stinging of the eyes. Twenty-six of these patients were women and 5 were men. Most of them were past 50 years of age. The rose bengal test was carried out and some had slight staining. A few of the patients were relieved by lubricating drops, and he feels that estrogen may be of help. While most of the discussions are dealing with tear secretions, Vouters [94]

discusses epiphora associated with normal nasolacrimal apparatus. He says that the sympathetic and parasympathetic fibers will stimulate the gland and produce epiphora. He says that psychic factors play a part, and he feels that the control of epiphora should be done on a physiological basis.

Canaliculi

Stern [95] thinks that we should always look for eyelashes in the puncta and discusses some of the means by which eyelashes get into the puncta. From my experience, lashes in the puncta are common and frequently give rise to epiphora. Stern thinks that the cilia are sucked into the canaliculus by the negative pressure in the lacrimal sac.

One of the most frequently missed inflammations of the eye is concretions in the canaliculus.

Moore [96] presents six cases in which actinomycosis was found. In one case he grew an organism which was called Actinomyces bovis Harz in 0.5% glucose agar shake medium. In one of his cases the canaliculus had eroded and involved the adjacent skin. From my own experience, in most of these cases there are diverticula of the canaliculi, in which the organisms become lodged and may invade the adjacent tissues. This may be one reason why Moore had to curette the canaliculus in a number of his cases. Nectoux and Suchet [97] apparently found Veillonella parvula in a canaliculus. They state that this organism is a Gram-negative coccus between 0.2 and 0.4 μ in diameter. It occurs in clusters similar to the Staphylococcus, but smaller in size. It is strictly anaerobic, and it was considered by Prevot (1933) to be a Gram-negative small Staphylococcus. They found two or three concretions in the canaliculus after tyrothricin had been used. No organisms could be grown from the canaliculus; however, they think that the tyrothricin made it impossible to grow the organism again after the original culture. It is interesting to note that I have seen an occasional concretion in the canaliculus, from which I was unable to demonstrate any Streptothrix or Actinomyces. I had assumed that the fungus had disappeared by natural means.

Most of the literature on the canaliculus deals with therapy on obstruction.

Henderson [98] published a follow-up report of five cases of repair of the canaliculus with polyethylene tubes for 14 to 38 months. He states that polyethylene tubes must be left in place in the canaliculus for 30 to 46 days. If it is removed in 10 days, the canaliculus usually becomes obstructed again. While he has used silkworm gut in the lumen of the polyethylene tubes, he feels that polyethylene tubing is the most satisfactory. My cases of using polyethylene tubing in the canaliculus have not been too satisfactory. Usually I have left the tube in for periods up to three months, and at the end of a year the canaliculus again closes. Veirs [99] uses nylon rods in the lower canaliculus and feels that three to four days is sufficient time for them to remain in place. However, he states that when he places the tubing in the nasolacrimal duct or through the lacrimal bone into the nose, the tubing is left in place for 90 days. His results are likewise gratifying.

When the puncta and canaliculus are everted and produce epiphora, Alekseyeff [100] uses electrocautery near the puncta on the conjunctiva. This simple method was used successfully in 62 patients. However, Ryaboshapko [101] cuts out a piece of the posterior wall of the canaliculus and keeps it open daily in order to prevent closure. This procedure was successful in 22 of 23 patients.

Etiology of Epiphora.—Alexsandrovitch [102] discusses 26 patients with epiphora and no demonstrable pathology of the lacrimal apparatus. He feels that in most cases the tearing is due to vitamin deficiency and that cod liver oil will eliminate it. Vitamin A may produce either hyperfunction or hypofunction of the lacrimal glands, he states.

Frieberg [103] feels that the lacrimal sac and canaliculi act as a suction and pressure pump to the conduction of tears. The caruncle transfers the pressure passively into the canaliculus when the eyes are closed; then, on opening the eyes, a suction is built up in the sac, which will actively pull the tears from the eyes.

Hudelo and Mergier [104] are not satisfied with the Schirmer test using filter paper and would replace it with strips of cigarette paper. They have developed an electric method to determine the pH of tears and have demonstrated that instillation of acid or alkali into the conjunctival sac is followed by a secondary adjustment of pH to the opposite range.

The causes of bloody tears were discussed by Meyer and Kluth.[105] They presented two patients; one had a capillary hemangioma of the conjunctiva and the other hysteria. They say that the diseases of the conjunctiva and of the lacrimal gland are also causes of bloody tears. In my experience, epidemic keratoconjunctivitis, gonorrheal conjunctivitis, and severe membranous conjunctivitis are the commonest causes of bloody tears. The authors also state that mechanical irritation, menstruation, and acetylcholine may produce bloody tears.

Epiphora associated with myxedema was discussed by Gilbert,[106] who thinks it is caused by edema of the face and eyelids combined with edema of the lacrimal ducts. In my experience it is due to the slight chemosis of the bulbar conjunctiva, which produces a fold over the puncta. The sucking action of the sac pulls the loose, edematous conjunctiva into the puncta and mechanically prevents tears from flowing into them. Chavarria-Lopez [107] reports a case in which a fold of the conjunctiva extended over the lid border, associated with marked epiphora. He excised the fold, with prompt relief of symptoms.

Lacrimal Sac

Raimondo [108] tries to explain the cause of chronic dacryocystitis on the basis of endocrine dysfunction. Since chronic dacryocystitis is common in middle-aged women, he tried the estrogens, without much effect. Garzino [109] examined 54 lacrimal sacs of subjects of various ages and found that in older people, particularly men, there is always a mild inflammatory reaction in and around the sac. There is also some sclerosis and scar formation. Whether the common findings of chronic dacryocystitis in older people are due to glandular dysfunction or to the increased prevalence of bacteria in the conjunctiva is not certain. I am inclined to believe that since older people are more likely to have chronic conjunctivitis with secondary dacryocystitis, obstruction of the nasolacrimal duct, with edema and later scarring, is a natural occurrence.

An unusual case of a calculus in the lacrimal sac is described by Weizenblatt.[110] It is apparently the first in the literature. The calculus measured 2 cm. in length and 7 mm. in diameter. It was undoubtedly inflammatory in origin, and Weizenblatt explains its formation as the result of a foreign substance forming a nucleus and the tears depositing calcium around it.

Tumors of the Lacrimal Sac.—Melanomas of the lacrimal sac must be extremely rare, since Eitrem [111] was able to find only three cases in addition to his own. His

patient was a 30-year-old woman with numerous pigmented nevi of the face. During an attempted dacryocystorhinostomy, a 5 mm. black area was discovered in the anterior wall of the sac. It was removed and found to be a benign melanoma.

In my experience, lymphomatous infiltrations into and around the sac associated with circulating leukemias are not uncommon. They produce all of the symptoms of chronic dacryocystitis but should not be attacked surgically, since x-rays not only will reduce the tumor, but will restore normal function of the lacrimal sac. Desvignes and Sadoughi [112] describe a case of what they called reticulolymphosarcoma of the lacrimal sac, but from their description I would call it a lymphocytic malignant lymphoma. The sac was removed, and histological examination aided in the diagnosis. The area was treated postoperatively with x-rays, without further extension of the tumor. I disagree with the authors, who state that microscopic examination is essential, since in my experience the clinical diagnosis is not too difficult and x-ray treatment is so gratifying.

Busse-Gravitz [113] feels that he can obtain valuable information with what he calls a dacryocytogram, in which he smears the tears and counts the cells. He states that there are four types of cells: the epithelial cells, two types of increasingly differentiating forms, and polynuclear cells. The more irritation of the sac, the more cells are found in the count. In inflammations there is a shift to the right with an increase in the number of adult polynuclear cells and an increase in small epithelial cells.

Robertson [114] reviews the treatment of dacryocystitis in infants and discusses the old controversy of conservative treatment versus probing. He feels that early probing gives the best results. Probing of the nasolacrimal duct is one of the most difficult procedures to teach in ophthalmology, and I am sure that in Robertson's hands probing is best for him, since he was able to cure 41 of 49 eyes with one probing. He states that no procedure gives such uniformly good results. Cusick and Michael [115] inject penicillin after probing, which they claim almost completely eliminates second probings. They also advocate injecting penicillin into the sac after removing the mucopurulent material. They use a long needle and inject through the skin.

Dacryocystorhinostomy.—Not only is dacryocystorhinostomy a popular operation, but nearly everyone who does it adds his own modifications. I have read more than 25 publications on this subject and numerous discussions. The operations can be divided roughly into four groups: the intranasal approach, probing of the nasolacrimal duct combined with insertion of polyethylene or similar substances; anastomosis of the sac to the nasal mucous membrane, and tunnelization of the nasolacrimal canal combined with a polyethylene splint.

Blankenstein [116] does the usual Dupuy-Dutemps procedure on infants and children. He thinks that there is no need to remove the sac of infants, even though it is hard to perform a dacryocystorhinostomy in such a small area. I share the feeling of Blankenstein and believe that a dacryocystorhinostomy, after unsuccessful probing, should always be done on infants or young children, in preference to any other form of surgery.

Jones and Boyden [117] think that the operation belongs to the rhinologists and says that the publication was presented "to stimulate interest among rhinologists in a surgical technique which they alone are best suited to perform and to describe a method of using the nasal wall of the lacrimal sac as a flap which, it is hoped, will

lessen the percentage of postoperative closures of the intranasal ostium." The intranasal operation is divided into three stages, preceded by a partial or complete submucous resection. The first stage is the preparation of the nasal mucosa flap, which consists chiefly of the mucosa from the middle turbinate. The second stage is the removal of the lacrimal bone, the area in the nose being marked by a special caliper and the thin bone drilled or cut through with a long drill or chisel. The third phase is the formation of the flap from the medial wall of the lacrimal sac. This flap is held in place by a Vaseline (petrolatum) gauze pack, while the pack is held in place by dermal sutures in the canaliculi. They report 38 cases, in 3 of which the intranasal window has closed postoperatively.

The intranasal approach is also advocated by Mirecki [118] and Jauerneck.[119] Mirecki performed 130 intranasal operations. Most of the time the operation was performed in two stages: A submucous resection is combined with a flap on the lateral wall of the nose, and, several weeks later, a large bony window is made through the lacrimal bone into the sac. Jauerneck's [119] procedure is similar.

There are numerous modifications of the Dupuy-Dutemps operation. One of the most interesting presentations was that by Tyrrell.[120] He tried to shorten the procedure in order to decrease the operating time. When there is not too much bleeding, he can do the operation in five minutes. He makes a large opening through the lacrimal bone with a chisel and extends the opening with ronguer forceps. He removes the mucous membrane of the nose and sac, leaving a hole. The only suture is for the skin closure. He begins irrigation on the second day, and his results are as good as any others reported.

Kaleff [121] claims to do the dacryocystorhinostomy without sutures. He makes a flap of the nasal mucous membrane and another flap of the sac. The sac flap is sutured to the skin, and then the skin is closed.

Norene,[122] Law,[123] Goldberg,[124] and Rolin [125] present slight modifications of the procedure. Norene's description of the technique of the operation is well presented and is worth reading by those not familiar with the technique.

Abrahamson and Stichel [126] favor the Stokes or the MacMillan procedure, which is modified by the use of steel wires placed into the nose in order to keep the ostium open, while Marin-Amat [127] uses horsehair between the sac and the nose to prevent closure of the ostium.

Summerskill [128] has reported extensively on the insertion of tubes into the nasolacrimal duct. He states that before tubes of polyethylene are inserted careful selection of cases must be made. Of all cases of dacryocystitis, he makes the following division:

1. Idiopathic dacryocystitis accounts for 76% of the cases. There is a definite hereditary tendency, the incidence being highest in women with a faulty development of the bony canal.

2. Infantile dacryocystitis accounts for 22% of the cases. Two per cent of these are not cured by probing and require dacryocystorhinostomy.

3. Acquired dacryocystitis accounts for only 2% of the total. They follow trauma, diseases of the sac, and tumors of the sac.

He has developed a 6 mm. polyethylene tube, which is sutured into the ostium between the sac and the middle meatus of the nose. He states, however, that polyethylene tubing of smaller size inserted through the nasolacrimal duct is successful in 60% of the patients.

Several ingenious methods of tunnelization have been devised. DeJean [129] made a pyramidal-shaped tube of acrylic resin which he places into the nasolacrimal canal. The tube is flanged at the top so that it will remain in the sac. The sac is opened in the usual way and a probe passed through the nasolacrimal duct. The duct is then cleaned out with a curette, and the bony portions are opened by means of a file. DeJean and Boudet [130] reported 30 cases treated by this method with excellent results. Roveda [131] and Wilde [132] also prefer to use a polyethylene splint.

Converse and Smith [133] describe methods of repair of the inner canthus after trauma and emphasize that dacryocystorhinostomy should be done while anchoring the medial canthus. They state that the repair of many of these injuries require the ingenuity of the surgeon. Repairs should be made at once if possible, since the canthus becomes rounded because of the pull of the orbicularis muscle. From my own experience with injuries of the face following automobile accidents, I can emphasize that when the sac is damaged it is best to perform a dacryocystorhinostomy at the time of the repair of the injury.

Lacrimal Gland

Diseases of the lacrimal gland are not too common, and Nover and Müller [134] believe that they have an explanation. They present the hypothesis, based on findings of Bautzman, that all exocrine glands have around their excretory systems a collection of lymphoreticular cells. These cells act as a defense mechanism against toxins and bacteria and form, together with the gland, the lymphoglandular system. They state that while lymphocytes are not present in the newborn infant, they develop in the adult. In babies a mucopolysaccharide present in the epithelial cells of the gland acts as the protective mechanism until the lymphocytes develop. Inflammations of the gland do occur, however, particularly in mumps, as was shown by Galpine and Walkowski.[135] Atrophy of the gland must occur in Sjögren's syndrome, and at times it may be associated with scleroderma, as was shown by Ercoli and Lepri.[136] Sabbadini [137] reported a granuloma of the lacrimal gland which he thought was noncaseous tuberculosis, but he presents little evidence to support his view.

Tumors of the Lacrimal Gland.—Mixed tumors of the lacrimal gland are common. Markey and Zelaski [138] were able to find 330 reported cases, and I know of a number which have never been reported. They think these tumors are indistinguishable from mixed tumors elsewhere in the body. They present a highly variable picture of epithelium and connective tissue. Steinmetz [139] believes there are two types: a true mixed tumor, which is extremely rare, and an epithelial neoplasm, which may or may not show myxomatous stroma. He states that recurrence even after extirpation is 100%.

The most interesting tumor is that reported by Rama.[140] A deeply pigmented mass was seen on the palpebral portion of the lacrimal gland. It was called "melanoma sarcomatodes of low degree of malignancy" by the author.

CONJUNCTIVA

Congenital Anomalies

Congenital defect of the conjunctiva is extremely rare. Epitarsus seems to be the commonest. Rabadan-Fernandez [141] reports a fold with a thick band in the conjunctiva extending from the outer canthus toward the cornea, associated with what he called a coloboma of the outer canthus. I think this must have been an epitarsal fold.

Cystinosis.—Since cystinosis is an inborn error in metabolism and is hereditary, I am including it with congenital anomalies. Cystinosis, according to Kennedy,[142] is a term applied to the particular form of cystine disease which occurs in infants and young children, as distinguished from the disease of younger children and adults. The latter form is characterized by the formation of calculi in the lower urinary tract, followed by secondary renal lesions. Cystinosis is characterized by deposits of cystine in various organs and tissues. Rarely, however, are renal calculi found. Cystinuria is probably the commonest of all inborn errors of metabolism, according to Kennedy,[142] and affects males twice as frequently as females. The metabolic block in cystinuria is a failure in the oxidation of cysteine to a sulfate. The accumulation of unoxidized cysteine causes its precursor, cystine, to accumulate in body fluid and spill in the urine. Cystine crystals are common in the conjunctiva, and biopsy of the conjunctiva is of diagnostic value to the pediatrician.

Guild, Walsh, and Hoover [143] report finding cystine crystals in the cornea but did not identify them in the conjunctiva. They feel that such crystals were probably overlooked and may have been present. Brændstrup [144] reports cystine crystals in the conjunctiva.

Normal Conjunctiva

Go [145] states that the anatomical construction of the capillaries in the limbus is the same as that in the skin papillae. These capillaries end in a bow shape, with one arterial and one to three venous shanks. He describes eight types of capillaries: continuous nongranular, wavy, intermittent nongranular, continuous granular, intermittent granular, bead-like (globular), static, and blood-free. He discusses the effect of 3% ethylmorphine hydrochloride (Dionin) on normal capillaries. Under ethylmorphine hydrochloride all capillaries dilate and contain blood. They are of the rapid, continuous, nongranular type. After two minutes, gray-white spots appear around the arterial shanks, which he thinks are exudation. After 30 minutes, the arterial shanks contract, the blood stream slows, and the capillaries become granular and sometimes static. After 60 minutes the capillaries become clearer and stasis disappears. They return to normal in 120 to 150 minutes. He states that the gray-white spots which develop around the arterial capillaries are 90% eosinophile leucocytes. The absorption mechanism acts through the venous shank while the arterial shank is constricted. He feels that the changes produced are caused by a direct action on the capillaries.

Arajärvi [146] examined premature infants to determine the sensitivity of their capillaries to epinephrine. He examined these infants before silver nitrate was instilled, since silver nitrate interferes with the observation. He found that 1:100,000 epinephrine hydrochloride constricted the capillaries in infants weighing 2,000 gm. or less. The full-term infants' capillaries were constricted by a 1:1,000,000 epinephrine solution. Therefore it requires a stronger solution of epinephrine to constrict the capillaries of the premature infants.

Landesman, Douglas, Dreishpoon, and Lee [147] describe the changes in the conjunctival vascular bed in normal pregnancy. They state that Kellogg and Hertig [148] found vascular changes in the terminal arterioles in toxemia of pregnancy. Landesman and associates [147] describe progressive vascular changes as a normal pregnancy proceeds. As the pregnancy progresses, the veins show a reduction in flow. From the third trimester on, and during labor, advanced changes occur in the arterioles.

and capillary reduction develops into tissue ischemia. True spasm of the vessels, mainly the arterioles, may develop and last for 48 hours post partum. As pregnancy develops there is an increased sensitivity of the vessels to epinephrine.

Friedenwald [149] has shown that the conjunctival epithelium from the limbus grows over the denuded cornea. At times it may carry some of the normal pigment with it and may produce the Stähli line. The conjunctival epithelium may be recognized by the presence of mucin droplets and the absence of glycogen. It takes about six weeks for the metaplasia of conjunctival to corneal epithelium to take place.

Binder [150] describes in detail the mucus cells of monkeys (Macacus rhesus). He thinks that they are derived from the basal cells and are the same as those that occur in man.

Bacteriology of the Conjunctiva.—Gilchrist Gibson [151] took preoperative cultures on 1,000 patients with normal conjunctival sacs. Of those with bacteria, staphylococci (Micrococcus) were found in 62%, one-fifth of which were coagulase-positive; Bacillus xerosis (Corynebacterium xerose) in 30%; Proteus vulgaris in 2.5%, and P. pyocyaneus (Pseudomonas aeruginosa) in 3.0%. He was not able to find a single diplobacillus or pneumococcus.

Schreck [152] reported on 200 Staphylococcus cultures obtained from the conjunctiva and found 9 were Staphylococcus albus, 1 of which was coagulase-positive and 8 coagulase-negative. He found hemolytic Staph. albus in 5 instances, which was coagulase-positive in 1 and coagulase-negative in 4; Staphylococcus aureus in 11, which was coagulase-positive in 3 and coagulase-negative in 8, and hemolytic Staph. aureus in 175, which was coagulase-positive in 51 and coagulase-negative in 124. Only 18 of the staphylococcic cultures were sensitive to sulfacetamide.

Conjunctivitis

Acute Conjunctivitis.—Tostevin [153] reports on acute conjunctivitis from Australia. Acute conjunctivitis, or pink-eye, has a duration of from 5 to 10 days. There is considerable discharge, edema, and small hemorrhages; however, the cornea remains clear. The common organisms are the Morax-Axenfeld bacillus, the Koch-Weeks bacillus, and the Pneumococcus; gonorrhea was so rare as to be not worthy of mention. His treatment consisted of penicillin, 1,000 units per cubic centimeter, or sulfacetamide, 10 to 30%, in which the pH was adjusted with chlorobutanol. He uses a 1:2,000 solution of methylrosaniline chloride (methyl violet) in the other eye. He says that zinc sulfate should never be used in acute conjunctivitis.

Davis and Hines [154] reports that Hemophilus aegypticus, or Koch-Weeks bacillus, and Hemophilus influenzae caused most of the conjunctivitis in elementary schools. The highest attack rate occurred in September, while the lowest was in December. The first three grades of school had more acute conjunctivitis than the other grades, and the white children were more likely to be affected than the colored.

Holmes [155] followed an epidemic of acute follicular conjunctivitis between January and June, 1951. He estimated there were 5,000 cases, or about 1% of the entire population. The onset was with redness, foreign body sensation, and some pain. There were many follicles on both tarsi and in the fornices, with multiple petechial hemorrhages, but no discharge. The patient began having slight discharge about the fifth day. There were no corneal changes and no evidence of a preauricular

lymph node swelling. Recurrences were common in from six to eight weeks, and repeated cultures produced nothing of note. This may have been an epidemic of Béal's conjunctivitis.

Egan [156] reports an interesting case of hyperacute conjunctivitis with suppuration of the preauricular lymph node. He found a hemolytic Staphylococcus in culture from the conjunctiva and many Gram-positive cocci on the smear. The conjunctivitis improved, but the preauricular lymph node had to be opened; it contained a yellow lumpy pus. Unfortunately, no culture was made of the pus from the preauricular lymph node. In the second eye a similar hyperacute conjunctivitis developed eight days later, with an enlarged preauricular lymph node. Chlortetracycline (Aureomycin) was given by mouth and prevented suppuration of the second lymph node.

Mitsui, Tanaka, and Hanabusa [157] report on the treatment of nine cases of acute bilateral conjunctivitis caused by Koch-Weeks bacillus. In one eye they used a combined oxytetracycline (Terramycin)-polymyxin B ophthalmic ointment, and in the other eye oxytetracycline-streptomycin ointment. The two ointments were equally effective.

Prophylaxis of Ophthalmia Neonatorum.—O'Brien [158] defines ophthalmia neonatorum as a definite purulent conjunctivitis of the newborn infant. It is not necessarily gonococcal, since gonorrhea, he states, is very rare in maternity units in England. Ophthalmia is a nuisance and the commonest form of neonatal minor sepsis. The incidence of conjunctivitis in infants varies from 1.5 to 6.0%. All of the male infants were treated with oxytetracycline drops or ointment, while the female infants were not treated but were used as controls. He stated that oxytetracycline is of no value in the prevention of ophthalmia. Simple cleansing of the eyelids is as effective as any form of topical prophylaxis.

Mallek, Spahn, and Mallek [159] compare the neonatal use of silver nitrate and penicillin in a total of 2,161 births; 1,073 infants received silver nitrate and 1,088 received penicillin ointment, in a concentration of 2,500 units per gram. A small group, of 180, received a solution containing 100,000 units per cubic centimeter as drops in the eye at birth. They believe that penicillin is a safe substitute for silver nitrate and that a method of administration should be selected which requires minimal handling of the infant's lid. Many infants had edema of the lids and red eyes after prophylaxis, possibly a few more with silver nitrate than with penicillin.

Martius [160] says that one-third of the infants treated with silver nitrate develop a conjunctivitis. He used an oily solution of penicillin, 100 units per gram, in 200 infants. Seven developed a slight conjunctivitis, which he called congestion, and one a catarrhal inflammation. He feels that the prophylaxis should be changed.

Herold [161] thinks that streptomycin-penicillin ointment is best for prophylaxis.

Holst [162] reports that 60 cases of blindness in Norway were due to blennorrhea neonatorum; however, the diagnosis was not certain in every case. It was of interest that not a single patient was under 20 years of age.

Epidemic Gonorrheal Conjunctivitis.—Raïs [163] reports that severe epidemic gonorrheal conjunctivitis, called "Rmad," is the main cause of blindness in North Africa; however, Hugonot [164] and Toulant, Larmande, and Toulant [165] contend that epidemic gonorrheal conjunctivitis is rather rare in French North Africa. Toulant and associates say that gonorrheal conjunctivitis occurs during August and

September and is not at all like the ophthalmia of the newborn. Both Hugonot and Toulant and associates state that the Koch-Weeks bacillus is the most frequent cause of purulent conjunctivitis in North Africa; particularly is it associated with central corneal ulcers.

Raïs [163] states that the epidemic conjunctivitis is improved within a few hours with chlortetracycline, and since this drug is so effective against trachoma, he feels that it should be used for prophylaxis. Toulant and associates think that 1% streptomycin is most effective against the Koch-Weeks infection.

Allergic Conjunctivitis.—Hanser, Moore, and Stickle [166] report on 572 conjunctival smears from cases of clinical ocular allergy. They prefer to scrape the conjunctiva rather than make direct smears. Slides were examined under a low-power lens to determine the per cent of eosinophiles. Cortisone solutions will decrease the number of eosinophiles.

Hanser [167] also reports an attempt to correlate the clinical findings with the per cent of eosinophiles found in the scraping. He states that if there is 3% or less of eosinophiles there are no symptoms, or the patients may be recovering from an attack. If there are from 3 to 10% of eosinophiles, then there are increasing symptoms; when there are 10% and above, the symptoms are severe. He states that cortisone used locally and Staphylococcus toxoid decrease the number of eosinophiles. He stresses history taking in order to make a diagnosis of ocular allergy.

Ruedemann [168] thinks that people are poorly cared for when they have an allergy and that doctors are many times responsible for sensitizing patients. As an example, he tells of a man who used butacaine (Butyn) in one eye until the vision was reduced considerably; later the patient got a foreign body in the other eye; butacaine was again used, and all economic vision was lost. Almost anything a patient comes in contact with can cause a local reaction. He thinks that face soap, hair washes, and cosmetics produce 1 to 2% of allergies. Recently beryllium has become widely used in industry; either the substance or the fumes of this material can cause a severe conjunctivitis. Ruedemann feels that in 90% of cases of ocular allergies a simple questioning will disclose the cause.

Payne [169] states that vernal conjunctivitis is a classical allergic disease of the eye. The pathology is mainly concerned with conjunctival smears, which contain many eosinophiles. Sections of the cobblestone plaques show thickened epithelium and papillary hypertrophy in the early phases, followed by fibrosis and signs of chronic inflammation. He feels that phlyctenular conjunctivitis and granulomas have an allergic phase; however, there is considerable lack of evidence for this. While chalazion may be considered allergic by some, eosinophiles are very sparse.

Prewitt [170] cites several interesting cases of various ocular allergies, some of which were due to house dust, wool, and the Staphylococcus. He stresses the importance of a careful history and prefers Hansel's [171] technique of high-dilution therapy.

One of the most interesting observations was made by Paggy,[172] who thinks that vernal conjunctivitis is caused by a sensitivity to one's own hormones. He was aided in his research by Zondek, who prepared the hormones. He treated 12 patients with injections of Astrophore, and one with testosterone propionate (Neo-Hombreol). At first the therapy made them worse; however, gradual improvement was seen. He stated that cortisone gave excellent results in four cases.

Theodore [173] tells how to obtain scrapings from the conjunctiva to aid in the diagnosis of allergic conjunctivitis. He discusses the physiology of the eosinophile and brings out that these cells are present in the blood in certain general diseases and are naturally linked with histamine. When the blood histamine is high in rabbits and in patients, they have a higher eosinophilia. A high blood eosinophilia does not increase the conjunctival eosinophiles. Theodore points out, with other workers, that severe chemical and vegetable irritants produce eosinophilia, and that one should be on the lookout for conjunctivitis from insect powder, lime, and other substances. He emphasizes an earlier observation of Bollack that eosinophilia may indicate a self-induced conjunctivitis. He feels, however, that most of these irritant types of conjunctivitis with eosinophiles produce different clinical pictures and are rarely confusing. In a true allergic conjunctivitis, eosinophiles may be delayed in appearing up to from 10 to 14 days, in the case of drug or cosmetic allergies. In vernal conjunctivitis, they vary from time to time. Generally there are many eosinophiles in vernal conjunctivitis. Many of the common drugs used in ophthalmology produce eosinophilia, particularly tetracaine (Pontocaine), butacaine, atropine, penicillin, and sulfonamides. He thinks there is an increase in basophiles during a remission of vernal catarrh and that the basophile may be the only cell suggestive of an allergy to scopolamine. Pilocarpine, physostigmine, and diisopropyl fluorophosphate generally do not produce eosinophiles or basophiles, as has been pointed out previously by Thygeson, but Theodore thinks these chemicals may occasionally produce a pure allergy with eosinophiles.

Theodore [174] classifies the types of allergic conjunctivitis as follows: 1. Acute allergic conjunctivitis. This is usually an atopic conjunctivitis with a family history of allergy. It is generally produced by inhalants, such as pollens, dust, danders, feathers, or food. There is an immediate allergic response to the substance; a positive intradermal test is obtained in from 15 to 30 minutes. The major characteristic finding in the conjunctiva is chemosis. 2. Allergic dermatoconjunctivitis. This is a delayed allergic response, requiring 24 to 48 hours to develop. A positive patch test will usually not develop in from one to five days. In most cases it is a contact allergy due to drugs, cosmetics, or apparel. The characteristic finding is eczema. 3. Chronic allergic conjunctivitis, of which there are two types—the atopic type, in which there is an immediate allergic response due primarily to inhalants, and the bacterial type. In the latter group, the allergic response is delayed and is produced by bacteria, fungi, and possibly viruses.

Theodore states that, although in most instances ocular drug intolerances are due to drug allergy, enough cases of drug irritation occur to make it an important factor. Some drugs, especially the alkaloids, may not in themselves be irritants, but they may deteriorate in solution to form degenerative products which may be irritative. The preparation of the buffer for the drugs should be carefully compounded to prevent these by-products from forming. He feels that it is important to differentiate between drug allergies and drug irritations.

Tuberculosis and Phlyctenular Conjunctivitis.—Riehm [175] presented a philosophical discussion on tuberculosis and allergies to tuberculin. He states that the allergy is one of specific fixation of the organisms. The increase in the size of the tubercle is due to allergy. During the bacteremia of general tuberculosis, abortive foci occur, and the bacteria disappear or are deposited at the periphery of the lesion.

When only small numbers of bacteria are deposited, then only a small amount of allergin is freed, but it spreads over a large area. Exudation is less prominent than proliferative organization.

Widmer [176] reviews 42 cases in which he attempted to show that conjunctivitis, mainly phlyctenular, had as a basis generalized tuberculosis. In four cases pulmonary tuberculosis developed, but apparently not from the previous conjunctivitis. Some of the patients were followed for long periods, up to six years, and there were no generalized relapses during this period.

Stankiewicz [177] states that there are frequent signs of inactive pulmonary lesions or active extrapulmonary lesions in scrofulous children; 95% of 74 children had positive skin tests.

Sobanski,[178] reporting in the same journal, who examined 177 patients, feels that a scrofula develops with tuberculosis because of the allergy to the bacillus, associated with a decreased resistance of the patient. He thinks that local inflammation or trauma may invite the allergy to develop in the eye.

Pagliarani [179] discusses the pathogenesis of tuberculosis of the conjunctiva and reviews three cases, in one of which the disease appeared to be primary.

Wheeler and Sinclair [180] think that both streptomycin and paraaminosalicylic acid or calciferol are valuable in the treatment of tuberculosis of the conjunctiva, while Zoelch [181] thinks that streptomycin is best. He treated 23 cases, with good results, and with relapses in 7. The treatment of scrofula is still subject to concern. All the above authors, including Plucinski,[182] discuss various forms of therapy. Harris' [183] discussion of cortisone is complete.

Phlyctenular ophthalmia, according to Magnus,[184] is an allergic reaction to some endogenous toxin to which the eye has become sensitized. It is most likely to be the toxin of the tubercle bacillus, but lack of food, fresh air, and cleanliness is an important factor. Heer [185] says that, in addition to tuberculosis, intestinal autointoxication is a factor and that toxic substances pass into the circulation because of liver damage. Pasino [186] describes a case with abnormal liver function.

Bocci,[187] in a series of experiments, attempted to sensitize the conjunctiva to killed tubercle bacilli by instilling an ointment containing 200 mg. of bacilli. Later, he [188] injected a similar material intravenously, and, after a period of 48 hours, conjunctival hyperemia developed, which he presumed to be a pure local allergy.

Parinaud's Syndrome.—Tularemia: Brini and Müller [189] discuss several causes of Parinaud's syndrome, which has been reviewed by Allen in January, 1953. An interesting outbreak of tularemia was reported by Thomas, Cordier, and Algan.[190] There were four cases of the oculoglandular type among 112 known cases of tularemia. Rabbits were the source of infection. The onset was a violent, acute conjunctivitis, with nodules and ulceration associated with preauricular lymphadenopathy and fever. Some of the preauricular glands suppurated.

Benign Infectious Lymphoreticulosis: The most interesting type of Parinaud's syndrome is called cat-scratch fever, or benign infectious lymphoreticulosis. Cassady and Culbertson [191] present an excellent review of the literature, stating that several organisms may produce the syndrome, i. e., Leptothrix, Myco. tuberculosis, Pasteurella tularemia, Sporotrichum, and viruses. They presented four cases of the oculoglandular syndrome, all of which showed positive skin tests with cat-scratch antigen, which had been supplied by Daniels and MacMurray. Daniels and MacMurray [192] made the antigen from a fluctuant axillary lymph node of a patient with a typical

history and findings of a cat-scratch fever. They state that "the antigen for intradermal testing is specific and free of false positive reactions. . . . A single negative test with a single antigen does not eliminate the possibility of the disease." They think the virus of cat-scratch fever belongs to the lymphogranuloma-psittacosis group of viruses.

French authors (Clément, Combes-Hamelle, and Meyer [193]; Lavergne, Thomas, Cordier, and Algan [194]; Thomas, Cordier, and Algan [195]; Sédan and Sédan-Bauby [196]) have reported extensively on cat-scratch fever. They have developed several antigens. It is interesting that in their reports they find positive skin tests in many members of one family. Clément and associates [193] report a case with involvement of epitrochlear and axillary lymph nodes in which all members of the family had positive skin tests. The cause of the disease is considered to be a virus, since cultures are bacteriologically sterile; however, I was unable to find any direct evidence that a virus had been demonstrated in the disease. Zarrabi [197] reports a peculiar follicular conjunctivitis with preauricular adenopathy in Teheran which resembles Parinaud's syndrome. Since he has seen numerous cases of it, and it is possible that these may belong to the group of cat-scratch fevers.

Ridley and Smith's [198] discussion of leptotrichosis conjunctivae is well prepared. They isolated a Leptothrix from a suppurating preauricular node. The organism differed from the one isolated by Verhoeff and King in that it did not ferment sugars and was strictly microaerophilic.

Virus and Rickettsial Diseases.—The virus and Rickettsial diseases should be divided into three groups, the first group comprising those in which the viruses have been grown and their physiology is understood, the second group consisting of those in which some of the characteristics of viruses are known to exist, and the third group those which are suspected of being virus diseases. Group 1 includes herpes simplex, vaccinia, lymphogranuloma venereum, psittacosis, lymphocytic choriomeningitis, influenza, mumps, yellow fever, Newcastle's disease, typhus, and Rocky Mountain spotted fever; Group 2 consists of herpes zoster, varicella, trachoma, inclusion conjunctivitis, rubella, measles, epidemic keratoconjunctivitis, rabies, common cold, dengue, sand-fly fever, Rift Valley fever, Colorado tick fever, infectious mononucleosis, infectious hepatitis, molluscum contagiosum, verruca, and foot-and-mouth disease, and Group 3, of benign lymphoreticulosis (cat-scratch fever), epidemic encephalitis, Mediterranean exanthematous fever, Rickettsial pox, tsutsugamushi fever, Q fever, recurrent fever, pemphigus, Reiter's syndrome, Behcet's syndrome, Stevens-Johnson syndrome, benign lymphogranulomatosis, malignant lymphogranuloma (Hodgkin's disease), and leukemia. (The foregoing is my division of viruses and Rickettsial diseases.) Cavarra and Bietti [59] published an excellent review covering nearly all the virus and Rickettsial diseases (Table).

Vaccinia: Most vaccinia lesions occur on the lids. However, Moffatt [199] reports an accidental autoinoculation of the conjunctiva. He states that 200 to 300 cases have been reported since 1796, with corneal involvement in 10 to 30%. When accidental infections with vaccinia are autoinoculations from a pustule elsewhere on the body, the prognosis is favorable; however, when the infection occurs from another person, the lesion may be severe. Marginal infiltrates of the cornea occur with vascularization; however, a true vaccinial pustule may occur on the conjunctiva and cornea. This type is usually serious. Moffatt also discusses the development of disciform keratitis.

Newcastle's Disease: Newcastle's disease of the conjunctiva was discussed in the 1953 review. Lippmann [200] states that the morbidity of Newcastle's disease in chickens is about 30%, and the mortality in young chickens is about 90%. He states that all human cases have had direct contact with chickens. He presents an interesting patient, whose left eye was infected in November and the virus was isolated. The patient recovered from this infection and the right eye became infected in December, but the second infection was milder. The virus is 680 mμ long, in sperm-like forms, or ovoid without a tail. He thinks that the serological diagnosis must be made with great caution.

Santoni and Bonaduce [201] were able to inoculate chicken eyes with the Newcastle virus, with the production of a systemic infection. Nelson, Pomeroy, Schrall, Park, and Lindeman [202] discuss the literature of Newcastle's infection since it was first reported, in 1943. They found 40 cases of conjunctivitis among 90 workers in a chicken-canning factory. Most patients had a unilateral conjunctivitis, and about half of them had a preauricular lymphadenitis. The authors report a serum neutralization titer in 64% of exposed workers who had not had conjunctivitis. Focosi and Scalfi [203] and Latte and Venturi [204] demonstrated that Newcastle's disease was infectious for rabbit eyes, but the virus did not appear to multiply.

Epidemic Keratoconjunctivitis: Apparently, sporadic epidemics of this disease are still occurring in areas around the world. Chang and Chang [205] report an interesting epidemic of 101 cases occurring in Peking; 61 were in males and 40 in females. They state that the incubation period is five days (Hogan and Crawford, 5 to 12 days; Thygeson, 7 to 10 days). Chang and Chang describe the typical picture of the disease: edema, follicular conjunctivitis, no discharge; in 73 of the 101 cases keratitis developed. Preauricular lymphadenopathy occurred in 77 cases. Many of the patients may have been contaminated in the clinic, since 13 staff persons had the disease. Another source which may have been related was the swimming pool in Peking. The authors feel that convalescent blood produced a favorable response. Pillat [206] observed 217 cases in Vienna in the fall of 1952, and in 55% the cornea showed opacities. He states that he was successful in transmitting the disease to rabbits by direct contact. In my experience, I have been unable to transmit the disease to the rabbit's conjunctiva or brain.

Arakawa and associates [207] isolated eight strains after serial blind passage in mouse brain. Two of the strains came from the conjunctiva and six from the serum of patients. The authors were able to demonstrate neutralizing antibodies in the patient's serum, but the titer was low when the keratitis was present. In my experience, the antibody titer is high so long as the corneal opacities are present. Brognoli [208] discusses a large epidemic in Pavia; he was unable to isolate the virus either in rats or in rabbits, using my technique. I have been able to isolate the virus in mouse brain only. Sezer [209] studied 25 patients and isolated the virus, on the chorioallantoic membrane of fertile chicken eggs, from four patients. After the virus had been maintained on the chorioallantoic membrane, it could be transmitted to adult mice by the intracerebral route. Before he inoculated the chorioallantoic membrane with the virus, he had planted grafts from human cornea on the membrane. The cornea-chorioallantoic membrane was then inoculated with scrapings from the conjunctiva. Serum neutralization tests on the virus were positive, indicating the presence of immune bodies. Sezer should be commended on the ingenious method

of isolation of the virus. I have heard by personal communication from Malaya and the Western Zone of Germany that strains of the virus have been isolated. I was unable to find any reports in the literature regarding this work.

Sanders [210] reviewed the old literature, and, in a personal communication, we feel that a conference of interested parties should be held.

Lepri [211] was unable to demonstrate intracytoplasmic or intranuclear inclusion bodies by phase microscopy in the conjunctival or corneal epithelium of epidemic keratoconjunctivitis. Thiel [212] states that epidemics are occurring in Heidelberg, Mannheim, and Central Franconia. He saw 80 patients with typical symptoms. While no treatment proved satisfactory, he [213] describes a new antibiotic called tyrosolvin which he thinks is effective. Tyrosolvin is tyrothricin and gramicidin made soluble in water by the addition of a quaternary ammonium base, cetyl pyridinium chloride.

McGavin, Boley, and Ormsby [214] followed outbreaks of epidemic keratoconjunctivitis beginning in March, 1951. The epidemics reached peaks, in number of cases, during the middle of April and in June. They followed 549 cases in four plants in Windsor, Ont. They feel that the disease is benign at the onset and gathers momentum, to become hyperacute at the height of the epidemic, and that as the epidemic terminates milder cases are seen. Corneal opacities were present in only 89 of the 549 patients, or 16.2%. Very few patients with mild symptoms develop corneal opacities. After one year, 29 of the 61 patients still had opacities; all complained of photophobia. Some of the patients without opacities (13) also complained of photophobia. No form of therapy was effective in the authors' hands. (No mention of convalescent plasma was made.)

Trachoma: Since there is considerable controversy regarding trachoma in the world-wide literature, I have depended on the World Health Organization report by the Expert Committee on Trachoma.[215] This Committee consists of G. B. Bietti, El Tobgy, Maxwell Lyons, Y. Mitsui, H. Moutinho, R. Nataf, R. Pages, and P. Thygeson. In their session in Geneva in March, 1952, they studied trachoma with a view to submitting "practical recommendations as to the possibility of successfully irradicating it by the application of modern methods of control, and to recommend effective, preventative measures against these diseases on an international level." It is the Committee's opinion that chlortetracycline and oxytetracycline are active against the disease, and they recognize that sulfonamides are likewise active, but that, because of the toxicity of sulfonamide, which of necessity must be given by mouth, it is not the drug of choice in mass therapy. They feel that for mass therapy chlortetracycline and oxytetracycline, 1% ointment four times daily, should be given without interruption for a period of two months. Resistant patients should be given sulfonamides. Surgery is considered as only complementary to medical therapy. They suggest methods of trachoma control in underdeveloped countries. They consider that trachoma is only slightly contagious and that immigration examinations should be continued, under competent ophthalmological service. They continue MacCallan's classification by further subdividing the classifications by the addition of a number of letters which would indicate the type of change in the conjunctiva, and of the complications, in words. For example, "Tr III + C + F V^s i^s trichiasis" means "trachoma in process of cicatrization with slight scarring and some follicles;

corneal vessels and corneal infiltration extending 5 and 4 mm., respectively, from the limbus; presence of trichiasis." They suggest international cooperation in research and present some problems for future research.

It is interesting to note that Funder [216] reports that in Vienna the number of cases of trachoma had decreased considerably; that has certainly been my experience in Iowa. The controversy in regard to therapy of trachoma continues, and there are those who oppose the treatment of trachoma by medical means. Of these, Diab and Abu-Jaudeh,[217] in Lebanon, state that chlortetracycline is of no value, and that sulfonamides, while they are effective against secondary infections, are not effective against trachoma.[218] Cat,[219] however, feels that chlortetracycline or oxytetracycline [220] acts more rapidly than other substances but accelerates scarring. However, he combines curettage with the medical therapy in all cases. In my experience, curettage produces considerable scarring. Mitsui [221] has found trachoma cured by oxytetracycline and chlortetracycline alone, as has been my experience. Jakober,[222] in Eastern Iran, thinks that Supronal combined with bandaging the eyes increases the effectiveness of the therapy.

Bietti,[223] Benigisu,[224] Victoria,[225] and Naccache [226] each consider the medical treatment as the most reliable.

Taborisky [227] presents a very interesting paper on the origin of trachoma; he feels that trachoma arose in Central Asia and that the tribes, as they migrated into Europe and Russia, brought trachoma with them. While there was little spread from these tribes to the local population, there may have been some. The Napoleonic War certainly spread the disease. The origin in Arabia is probably from Asia also, with spread into North Africa and Spain. Many of the Jews had trachoma, and they brought it to certain areas, particularly Amsterdam, Netherlands. From my own experience, it was common among Jews in New York.

I am not convinced that Mitsui, Tsutsui, and Tanaka [228] necessarily photographed the virus of trachoma. The bodies differ in size, and the epithelium contains so many mitochondria granules that I am not sure whether or not I am looking at the virus in the photographs. Kall [229] claims to have transmitted the trachomatous material to mice.

While most of the surgery for entropion and trichiasis of trachoma has been discussed under "The Lids," it may be noted that Putchkovskaya [230] uses mucous membrane grafts for the symblepharon. The surgical treatment of entropion and trichiasis is varied. Laborne,[231] Rappaport,[232] and Romanes [233] each suggest forms of surgical procedures.

Inclusion Conjunctivitis: Ormsby, Thompson, Cousineau, Lloyd, and Hassard [234] carried out extensive investigations on the etiology of ophthalmia neonatorum and studied 97 babies with conjunctivitis in a series of 1,703 births. Of these, they found seven with inclusion conjunctivitis. There was little difference in the rates of recovery with sulfacetamide, chlortetracycline, and oxytetracycline (Terramycin). However, they present the cases of several adults in which symptoms were suppressed but the recurrence rate was high. In four cases of chronic inclusion conjunctivitis, no inclusions were found on first examination, but after cortisone therapy many inclusion bodies could be found. The following is a list of all the organisms they found in 97 babies: Staphylococcus pyogenes, 47; Diplococcus

pneumoniae, 11; a Streptococcus of the viridans group, 11; inclusion bodies, 7; Aerobacter aerogenes (Bacillus lacti aerogenes), 4; Neisseria gonorrheae, 3; negative (no growth), 14.

Unusual Forms of Conjunctivitis.—Hudson [235] describes five cases of hemochromatosis with pigmentation of the skin, cirrhosis of the liver, diabetes mellitus, and hypogonadism. All cases were proved by liver biopsy. Two of the patients had diabetes mellitus; two had a prediabetic blood sugar curve, and one had a normal glucose tolerance curve. Four of the five patients had conjunctival microaneurysms, while one had retinal microaneurysms. In our experience in the University of Iowa Hospital clinic, microaneurysms in the conjunctiva are a common finding, particularly in debilitated patients.

Theodorides and Koutrolikos [236] report what they call blastomycosis of the conjunctiva. They show a white spot on the bulbar conjunctiva in the palpebral fissure. From the photographs it looks like a Bitot spot. While they were unable to grow the organism, fresh preparations showed the fungus. The material presented is not very convincing. Yourish [237] reports two cases of Bitot's spots without vitamin A deficiency. We should be on the lookout for blastomycosis, resembling Bitot spots.

Geserick [238] saw 18 patients with conjunctivitis caused by the hair of Dendrolimus pini (caterpillar). He states that the hair is not visible at first, but that the eye is irritated, with local thickening of the conjunctiva. When linear scratches appear on the cornea, the hair becomes visible and can be extracted. In this fascinating discussion, the wanderings of the hairs, Geserick thinks, are due to a hook on the sharp end of each hair, which helps to propel the hair, in association with the inflammatory reaction around it.

Jones [239] describes a patient with conjunctivitis from whose eye the larva of Oestrus ovis was removed. This larva is sometimes found in the paranasal sinuses of sheep.

Paufique and Moreau [240] describe three cases and review the world-known literature of 15 cases of "woody" conjunctivitis. The disease usually occurs between the ages of 2 and 6 years, and there is a familial tendency. It begins with nasopharyngeal infection and a membranous sore throat. The vulvovaginal mucous membrane is also involved. There are three cardinal signs: a pseudomembrane on the palpebral conjunctiva, yellowish white and 1 to 2 mm. thick; wood-like induration of the lids, and corneal change, which leads to perforation. The authors describe the fibrinous membrane over and in the conjunctiva as keratinization and areas of deep necrosis with crypts filled with pseudomembranous exudate. There is also a severe fibrinous reaction of the lids with the induration, which leads to the "woodiness." They state that beta irradiation is the only worth-while therapy. Winter and Michler [241] report two cases of membranous conjunctivitis of unknown origin in white brothers, aged 1 and 4 years. The authors consistently found pneumococci Types XIX and VII. In reading their description, I was reminded of Paufique and Moreau's patients.

Hogan [242] saw five cases of atopic eczema with bilateral keratoconjunctivitis. He felt that the changes were so typical that the entity deserved the specific title of atopic keratoconjunctivitis. The atopic patient is believed to become sensitized during infancy. It is an immediate reaction, and the sensitizing agents, called atopens, are

numerous. The conjunctivitis is associated with burning and mucoid secretion; remissions and exacerbations are common. Secondary, staphylococcic infections occur. The keratitis is typical. There is prompt response to cortisone.

Kornblueth, Laufer, Loebel, and Frisher [243] report a case with yellow masses in the conjunctiva which they studied histologically. They thought the lesion might be a variant of a plasmoma but described it as fibrinoid degeneration of collagen. The masses were easily removed from the conjunctival tarsus. Chinaglia [244] describes a similar change which he calls paramyloidosis, but it occurred on the bulbar conjunctiva. He thought that it was produced by a local irritation.

An interesting historical and clinical discussion of the eye in relation to gout is presented by McWilliams.[245] He describes what Hutchinson called the "hot eye of gout." The conjunctival vessels are dilated and tortuous. The eyes burn and are hot, with a prickling sensation. The symptoms change with the weather and the diet of the patient. Biopsy of a lesion at the limbus showed it loaded with needle-like crystals, characteristic of uric acid.

Kennedy [246] found 12 patients with keratoconjunctivitis and generalized leprosy. Klaude,† in discussing Kennedy's paper, says that ocular manifestations of leprosy are almost always present in the nodular form. Harley † states that while 90% of lepers have ocular involvement, conjunctival lesions are uncommon.

Croll and Croll [247] discuss two well-documented cases of trichinosis confirmed by biopsy. They stress that the ophthalmologist may be the first to see the patient because of the extreme acute swelling of the lids and conjunctiva.

A case of hypercalcemia with nodules on the conjunctiva, due to hyperparathyroidism, is described by Galindez-Iglesias and Lafuente.[248] Tricalcium phosphate was found in the conjunctival nodules.

A case of Bazin's hydroa vacciniforme is presented by Chinaglia,[249] who claims that the conjunctival changes are similar to those found in vernal conjunctivitis. There was also extensive scarring of the cornea.

Birge's [250] review of mycotic infections should be read by every ophthalmologist. He feels that mycotic infections are frequently missed and, like Fazakas,[41] wishes to emphasize their importance on the lids, conjunctiva, and lacrimal apparatus.

The ocular involvement in onchocerciasis varies in location, according to Sarkies.[251] About 35% of the patients show some ocular involvement. The corneal opacities are so typical that it is not necessary to demonstrate the filaria in the conjunctiva. The diagnosis is made by taking snips of the bulbar conjunctiva, mounting them in saline, and examining them immediately for the presence of the microfilariae. Hughes and Daly examined 32 cases and found filariae in 21 (cited by Sarkies).

The ocular manifestations in infectious mononucleosis are discussed by Jones, Howie, and Wilson.[252] Ophthalmic problems arose in 103 cases, in 84 of which there was acute conjunctivitis. They feel that the conjunctivitis resembles that described by Béal as acute follicular conjunctivitis. The conjunctival exudates contain small and large lymphocytes with abnormal large basophilic "infectious mononucleosis cells." Conjunctival biopsy findings are characteristic of infectious mononucleosis. Secondary infections also occur.

Vrabec [253] reports on the conjunctival changes of cutaneous psoriasis. Pillat, cited by Vrabec,[253] found only five cases in the literature and would accept only one

† In discussion on Kennedy.[246]

of these. Vrabec's patient was a 36-year-old man with psoriasis. The left eye had been in poor condition for some time. There were multiple plaques on the conjunctiva. He had had psoriasis lesions on the cheek, and at times on the upper lid. The plaque on the conjunctiva was 12 mm. from the limbus and 1.5 mm. in diameter. The cornea in the same eye had a triangular opacity, with the base at the limbus and the tip toward the pupil. The surface was irregular and wrinkled. Histologically, the conjunctiva showed lymphocytic infiltration over a wide area. The lymphocytic infiltration occurred in two planes: one in the basal layer of the epithelium and the other around the vessels of the corium. The epithelium was thickened and showed some changes and hydrops. There was infiltration of polymorphonuclear cells, but no lymphocytes, in the epithelium. The center of the plaque stuck out. The superficial cells were flattened and there was no true keratinization, but parakeratosis was present. There were suggestions of superficial scales but they were not numerous, and I thought this was because of the tears. In the granular layer of the epithelium there was evidence of keratosis, and the author called the granules inclusions.

Kall [254] reports on further observations of the protozoal conjunctivitis in Denmark. It has been previously reported in Allen's 1953 review; the present report is in addition. Kall was able to transfer these bodies in the red blood cells to mice and found them in the lungs and mediastinal arteries. He found these bodies in the peripheral blood of three patients with conjunctivitis and upper respiratory infection. The bodies appeared to pass through several stages. They are usually red and may be free, and some take on a serpentine form.

Sjögren's Syndrome: Morgan and Raven [255] present a beautiful discussion of Sjögren's disease for the general physician. Most ophthalmologists are well acquainted with this disease and have discussed it for some time. Morgan and Raven, however, emphasize the nonocular symptoms, which a few of us have discussed before. They stress biopsy of the parotid as an aid in diagnosis. Their review of the entire literature is excellent. In the discussion of the etiology of the disease, they consider five possible factors: 1. The endocrine basis. This theory is inviting, since the disease is commoner in females who have peculiar physical characteristics. They found a number of cases reported in which improvement followed stilbestrol, estradiol benzoate (Progynon B), and progesterone. It is so rare in males that it is difficult to diagnose, but estrogens may be a factor in the development of the disease in males. 2. Avitaminosis. This theory has been largely abandoned because of little evidence to show that the patients have vitamin deficiency. 3. Chronic infection. There has been much discussion of bacteria and bacterial toxin producing the disease. There is also some evidence that bacterial hypersensitivities may be of importance. However, both the bacterial and the virus theories are not very convincing. 4. Neurotopic factor. This theory has been fostered by Sjögren and his group, but Morgan and Raven are not convinced. 5. Heredity. This may be a factor; old rheumatic fever and adrenal insufficiency are also considered as of some importance.

The clinical findings may be presented as the ocular and the nonocular manifestations. The ocular manifestations are well known to most ophthalmologists. However, the Schirmer test and the rose bengal test should be stressed. Of the nonocular manifestations, the most important are dry mouth (xerostomia) and parotid swelling. The swelling of the parotid was first stressed by Bruce in 1941.

Arthritis of the hands is typical. There is marked dryness of the skin and mucous membranes and absence of perspiration, and the tongue is atrophic. The dryness of the vagina and loss of hair, particularly in the region of the vulva, may suggest hypothyroidism. The dental changes are advanced, and most of the patients have lost many of their teeth. In the digestive tract, achlorhydria and achylia are important. I have found that there is no hydrochloric acid in the stomach, even after histamine.† Constipation is troublesome, and the patients usually have gastric crises, which I think are similar to those occurring with pernicious anemia.‡ The blood changes in Sjögren's disease are variable; however, most patients have a macrocytic hypochromic anemia. Some, however, may have microcytic anemia. They also have low blood pressure. Telangiectasis of the lips and finger tips is not uncommon, and acrocyanosis may be present.

I have found the central nervous system symptoms to be rather marked. Morgan and Raven stress the mental changes that occur in these patients, who sometimes have epileptic fits. The pathology of the parotid glands is that of typical round-cell infiltration with atrophy and fibrosis. They state that it is more like that of Mikulicz' disease.

Poli and Wiel [256] report three cases which they felt were due to a pararheumatic virus. They discuss the stimulation of the sympathetic and parasympathetic nervous system to improve the glandular secretion.

Reader, White, and Elmes [257] report performing Schirmer tests on a series of 47 women and 15 men with rheumatoid arthritis, and the results were compared with those for 47 women and 15 men with nonrheumatic illnesses. None of the patients had ocular symptoms. There were, however, significant differences in the tear function between the two groups. Those with rheumatoid arthritis showed decreased tear function, and when the figure was compared with that for the normal series the accuracy was $P < 0.01$. They did an autopsy on a 28-year-old woman, who was thought to have had Sjögren's disease for 10 years, with the eye symptoms, dry mouth, and parotid swelling. Her rheumatoid arthritis was mild; however, during life she had acute gastric enteritis with fever and died in one of these attacks. The right lacrimal gland showed chronic inflammatory change and moderate lymphocyte and plasma-cell infiltration. Many of the acini were disorganized, and some of the ducts contained inspissated material. There was advanced fibrosis in the gland. There were renal lesions and gastric lesions, which they were unable to explain.

Angus,[258] at a meeting of the Victorian Branch of the British Medical Association, reported on a patient with Sjögren's syndrome. He thought that both the arthritis and the eye manifestations were part of a general collagen disease. He showed that the pathology was essentially chronic edema followed by hydropic degeneration and atrophy of the epithelium of the lacrimal glands.

O'Day [259] wishes to stress the use of the rose bengal test instead of the Schirmer test as an aid to the diagnosis of Sjögren's disease. He uses 1% rose bengal-chlorobutanol (Chloretone) water and feels that early cases of dry eyes can be demonstrated.

There is considerable discussion of the treatment of Sjögren's disease, and most of it relates to use of corticotropin and cortisone. Lepri [260] found that local treat-

‡ "Ocular Findings Frequently Diagnostic of Some General Diseases," read at a meeting of the Pacific Coast Oto-Ophthalmological Society.

ment with cortisone combined with its systemic use, or its systemic use alone, was of value in cases of Sjögren's disease. The subjective symptoms improved, and in one case he was able to demonstrate a reduction in the dryness of the mouth; however, the effect was not lasting.

Codman and Robertson [261] report variable results with corticotropin therapy in two cases of Sjögren's disease. The parotid swelling decreased; however, the eye and mouth symptoms were not changed.

Sjögren and Eriksen [262] gave up to 510 mg. of corticotropin and were unable to show any influence on the tear function in four cases. Coste, Offret, Faulong, and Uro [263] report no effect with cortisone solution in the eyes and very little on the general polyarthritis. From my own experience, cortisone and corticotropin are of little value except to improve the patient's feeling of well-being.

Pemphigus: Since pemphigus is so closely allied to Stevens-Johnson disease, and so is of concern to the ophthalmologist, I felt that he should have some understanding of the disease.

Lener [264] presents an intensive study of all forms of pemphigus, with considerable clinical and pathologic data. He states that since 1935 the following classification has been used: pemphigus vulgaris acutus (malignant pemphigus vulgaris); pemphigus vulgaris chronicus (benign pemphigus vulgaris); pemphigus vegetans; pemphigus foliaceus; pemphigus erythematosus (Senear-Usher syndrome); benign mucous membrane pemphigus (pemphigus conjunctivae).

Pemphigus vulgaris acutus. The bullae on the skin are flaccid and small in size. They break easily. There is no tendency for them to heal except by verrucous elevation and vegetations. There are extensive oral lesions, and oral lesions may develop first. They may be limited to the mouth for several months before the other skin changes occur. A large percent of these patients are Jewish. Histologically, the skin shows intracellular edema with loss of the intra-epithelial-cell bridges. There are marked changes in the basal epithelium cells, and they develop subbasal clefts. The bullae develop next, between the basal layer and the rest of the epidermis. The amount of detachment of the epithelial cells varies greatly. Eosinophiles are few when papillomatosis is pronounced.

Pemphigus vulgaris chronicus. Clinically there are large, tense bullae of considerable size, which have a tendency to heal. The mucous membrane in this disease is singularly free. The amount of involvement of the mucous membrane is a good clinical differentiating point between the chronic form and the acute form. There are very few Jews in this group. The prognosis in the chronic form is good except in elderly people. However, recurrences and exacerbations are common. The differentiation from erythema multiforme may be very difficult, and Goodman, cited by Lener, regards pemphigus vulgaris chronicus as a variant of dermatitis herpetiformis. Histologically, there are some subepithelial vacuoles with only slight intracellular edema. The intracellular bridges are not damaged and may be stretched. Most of the bullae are entirely subepithelial, but may be subepithelial in the center and intra-epithelial at the periphery. Eosinophiles are often numerous and may be the only evidence of inflammation in the bulla.

Pemphigus vegetans. This is a variant of acute pemphigus vulgaris; however, in this disease the bullae first grow into large areas, which become denuded, and the verrucous vegetations are numerous. This disease is invariably fatal. Histologically,

there are striking findings, in which intraepithelial abscesses occur composed almost entirely of eosinophiles. The abscesses are just above the basal layer. Epithelial hyperplasia is present, particularly around the eosinophilic abscesses.

Pemphigus foliaceus. In this disease, redness and swelling are present from the beginning, not only around the bullae but in areas without bullae. The lesions usually begin on the face, chest, and upper back, and oral lesions are rarely present. The prognosis depends upon the age of the patient; older patients die, and younger ones live. Histologically, there is acantholysis of the upper epidermis, usually in the granular layer. The acantholysis is present not only in the roof but in the floor of the bulla. The horny layers above the bulla are usually involved. The granular layers of the epithelium are deeply basophilic, resembling keratosis follicularis. Many inflammatory cells are present, including some eosinophiles.

Pemphigus erythematosus. The diagnosis of this disease is not entirely clear, but the condition apparently is similar to seborrheic dermatitis or lupus erythematosus, which progresses to pemphigus vulgaris and usually ends fatally. Lener [264] had trouble in making the diagnosis, but he found one case that was like pemphigus foliaceus.

Benign mucous membrane pemphigus (pemphigus of the conjunctiva). About one-half of these patients show some skin changes. The conjunctiva is most likely to be effected, showing shrinkage and scarring. The oral mucous membrane is next most commonly involved. Death is rare; however, obstruction of the esophagus may be very severe. None of these patients were Jewish. In my own experience, many were of Italian extraction. Histological study shows subepidermal bullae. There are no degenerative changes, and there is considerable inflammatory response and edema in the upper corium. Acantholysis was not present.

Tumor of the Conjunctiva.—Kennedy and Sullivan [265] think that epithelial plaques on the conjunctiva are precancerous lesions, although in one of their patients the condition histologically looked like Bowen's disease (carcinoma in situ). They concur with the findings of Ash and Wilder (1942).

Hereditary hemorrhagic telangiectasis is not uncommon, according to Miles.[266] He states that 175 families, involving over 1,000 members, are reported in the literature. Blood spots develop on the conjunctiva; these spots become larger with age, and hemorrhages become commoner. Bright-red, violaceous, or purplish lesions are found on the face, lips, tongue, conjunctiva, finger tips, etc. He found pinhead-sized lesions on the upper tarsus and a few on the lower lid. The patient had a daughter with a similar condition and another daughter without the disease.

Reese [267] gave an interesting paper in Sydney, Australia, on melanosis of the conjunctiva. He stated that conjunctival nevi in children are rarely malignant and about 30% were nonpigmented. The nevi are ordinarily salmon in color and usually at the limbus. Melanin may increase rapidly, especially at puberty, and the lesion may appear to grow. Malignant growths show increase in size of the nutrient vessels. All nevi should be excised, according to Reese, while for malignant nevi anything short of exenteration increases the mortality. He states that melanosis may be congenital or acquired. It may be within or beneath the conjunctiva. Spider-shaped stromal melanoblasts can be seen with the slit lamp. The incidence is about 12%. They never become malignant. Conjunctival melanosis occurs in the basal layers of the epithelium in a mottled area near the limbus. It is difficult to determine its malignant status. Acquired melanosis is usually precancerous. It is frequently

first seen in adult middle life. The lesions are nonelevated and irregularly shaped with variable density of pigmentation. The elevations are unusual even in the malignant phase. Mortality is extremely high. While Reese feels that an accurate differential diagnosis can be made, Dennis [268] thinks the differentiation of benign and malignant nevi is very difficult. Dennis feels that trauma or superimposed conjunctivitis can cause a nevus to become malignant.

Lymphomatous infiltration of the conjunctiva is discussed by Cometta,[269] in which he had difficulty in making the diagnosis. Duc [270] describes a patient with lymphomatous infiltration in the conjunctiva associated with lymphoid leukemia. Lymphoma of the conjunctiva and adnexa is common, in my opinion. They occur with or without circulating leukemias. The ocular changes in Hodgkin's disease have been extremely rare. However, an interesting infiltration of the bulbar conjunctiva occurring in a patient with Hodgkin's disease was described by Quintieri.[271]

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Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Willis S. Knighton, M.D., *Chairman*

Raymond E. Meek, M.D., *Secretary*

Feb. 16, 1953

Extraction of Complicated Cataracts. Dr. John M. McLean.

Complications of cataract surgery were discussed, starting with some of the complicating factors which are present before the operation begins and some of the author's views on how they should be managed. The most stimulating new development in cataract surgery in many years is Ridley's recent work on the intraocular implantation of the acrylic lens. It is felt that, while this method is deserving of study, it has not yet been properly evaluated and is not recommended at the present time.

Variations in preoperative medication, anesthetics, and preparation in the face of complicating situations were discussed. General anesthesia with thiopental or general akinesia with curare was recommended for unruly or apprehensive patients and those in whom communication is difficult through language difficulty or impaired hearing.

Shallow sections widened with scissors or ab externo incisions were advocated for shallow or abolished chambers. Deepening of such chambers to allow ordinary sections was given as an alternate. Purely corneal incisions were recommended for patients with bleeding tendencies. Deep scleral sections were advised to avoid exacerbation of a preexisting Fuchs's dystrophy. Anterior, temporal, and inferior incisions were offered in the face of functioning glaucoma filtration, although section through the bleb was suggested for eyes with hypotony.

The indications for iridotomies and iridectomies were then considered in detail, after which delivery of the lens was discussed.

A complicating factor considered is a lens which must be extracted in the face of an incipient uveitis; this operation has been done with fair success by intensive preoperative treatment with corticotropin, which is continued through the convalescent period.

The author stated that it was his considered opinion that fluid vitreous, high myopia, and old uveitis are not contraindications to intracapsular extraction, but are positive indications for that type of lens removal.

Contact Lenses in Aphakia. Dr. Elizabeth F. Constantine (by invitation).

The proper handling of a monocular cataract with a normal, or nearly normal, second eye has always posed a problem to the ophthalmologist. In the absence of complicating factors, such as subluxation, hypermaturity, or glaucoma, it often seems advisable not to intervene surgically. The advantage of the increased peripheral vision which is gained by removal of the cataract is frequently offset by complaints of dizziness, diplopia, nausea, etc. Above all, it is very difficult for the patient to realize that he has gained anything by submitting to the operation or to understand why he cannot be fitted with spectacle lenses for both eyes.

In an attempt to deal more successfully with this type of case, Ridley developed his intraocular plastic implant. The inherent dangers of his procedure are familiar to all, and the majority of ophthalmologists feel that its use is therefore not justified at the present time. This study was undertaken in an effort to offer an alternative whereby such patients could recover binocular vision with greater safety. It is far from complete, and this paper is presented only as a preliminary report in the hope that it may prove suggestive and encourage further work in this field.

The primary reason for inability to obtain binocular vision in monocular aphakia is, of course, the aniseikonia produced by the high anisometropia. A contact lens, because its position is much closer to the focal point of the eye than that of a spec-

tacle lens, reduces this to a minimum. Its possible value in such cases has long been considered, but it does not seem to have been widely used. Our object is to determine the proportion of patients who can, in the first place, achieve binocular vision by this means, and, second, wear such a lens successfully.

We have to date fitted 28 cases of monocular aphakia with contact lenses. Of these, 5 are regarded as having complete fusion, 14 as having incomplete fusion and are still being followed, and 9 have decided to give up their lenses after a brief trial, mainly because the idea of wearing the lens made them, in their own words, "too nervous." A point of interest here is that seven of the nine who reneged were clinic patients. As a group, they were of definitely lower intelligence than the group which persevered with their lenses.

Of the 28 patients, 3 gave up their lenses before any fusion tests were done, and 5 others had poor vision or a manifest squint, in which there was no hope of obtaining fusion without further surgery.

We have, therefore, 20 cases in which fusion tests have been done and in which we hoped to obtain binocular vision. All these patients had 20/30 vision or better in the eye not operated on and obtained equally good vision in the aphakic eye when fitted with a lens. One of these (5%) obtained binocular vision with the first trial of the lens. Seven others achieved it after a few weeks of wearing their lenses. Nine others were able to fuse with correcting prisms, and while five of these ceased to report, the other four have remained under observation.

We are attempting to develop fusion in these, as we did in the seven successful cases, by one of two devices or by both. One is occlusion of the phakic eye, in order to exercise the aphakic eye. The other is the use of temporary prisms to correct the muscle imbalance. When fusion is established with these correcting prisms, their power is gradually reduced.

The 3 remaining cases in the series of 20 have not been seen since they were first given their lenses.

There are, then, 8 patients who are able to fuse, or 40% of those we considered as possibilities, and 32% of the total number in which fusion tests were done.

In evaluating fusion, the red glass, Maddox rod, and Worth dot tests were used. Subjective diplopia was invariably present at first in all the patients who later established fusion, but it disappeared rather early, while the red glass test became negative next, and then the Worth dot test. The Maddox rod test usually will show some deviation in any case. In the figures just given, the Worth dot test was used as the criterion of fusion.

In addition to the cases of monocular aphakia, we have fitted a number of cases of binocular aphakia with contact lenses—some with the corneal type, some with the Lacrilens, and some with conventional molded lenses. In these cases, while the reduced image size is of value, the other advantages over spectacle lenses come into play. They are the increased field of vision, the removal of distortion due to spherical aberration, the elimination of astigmatism, and the much lighter weight. Here, too, as in other spheres of contact lens use, there are the occupational advantages in certain fields where spectacles are a detriment. The disadvantages are the annoyance, irritation, and sometimes inability to tolerate the contact lens. Of our 19 monocular patients wearing lenses, 8 wear theirs full time—all day long. Two wear theirs about half a day; three others only approximately two hours at a time, and the status in this respect of the remaining six is not known at the moment.

While we have no specific figures on the wearing time in our cases of binocular aphakia, there is one case I should like to cite. This patient was fitted some time ago with conventional molded lenses. She wore them all day, every day, for a number of years, with complete satisfaction. Finally, she died, of carcinoma—not of the eye—and in due course came to autopsy, still wearing her contact lenses. This threw the pathologists into a state of utter confusion, which was only resolved by an ophthalmological consultant.

We have used the corneal type of contact lens in this series of cases, not because we feel that it is superior to any other type. Its chief advantage is that it permits

trial of a contact lens by the patient with the least financial investment, being a stock lens and therefore returnable. In cases in which the patient obtains binocular vision but is not able to tolerate a corneal lens, it is possible that another type of contact lens may prove satisfactory.

Another definite advantage of the corneal lens is that it can be used in cases in which muscle surgery has been done or is contemplated, where the resulting scleral irregularity often precludes fitting other types of contact lenses.

In summary, we have shown that it is possible in some cases of monocular aphakia to obtain binocular vision by means of a contact lens. The results in this small series, while far from conclusive, seem to us to warrant further investigation, and we look forward with interest to other reports on the subject.

Complications in Cataract Extraction. Dr. Jack S. Guyton (by invitation), Baltimore.

Vitreous loss, or incarceration of vitreous within the incision, is the most important operative complication of cataract extraction. The vitreous normally consists of a framework of insoluble fibrillae, arranged in an onion-peel, sheet-like fashion, permeated by a somewhat viscous fluid. (The structure of the vitreous and measures to prevent loss of vitreous were then discussed in detail. For retrobulbar injection 2.0 cc. of 4% lidocaine [Zylocaine], hydrochloride containing epinephrine hydrochloride, 1 : 20,000, with or without hyaluronidase, was recommended, regardless of high blood pressure or heart disease. A moving picture illustrated vitreous loss during operation.)

After vitreous loss there may be (1) interference with the dehydration mechanism of the cornea sufficient to cause corneal edema, (2) vitreous opacities, (3) an increased incidence of retinal detachments, (4) an increase in postoperative uveitis, and (5) an increase in postoperative glaucoma.

In the handling of vitreous loss at the time of operation, the incision, of course, should be closed with as little loss of vitreous as possible; then, if the incision does not appear to be tightly closed, additional sutures should be inserted. The injection of air, together with instrumental freeing of vitreous strands from the incision after the sutures are tied, has been disappointing in my hands.

DISCUSSION ON PAPERS ON CATARACT EXTRACTION

Dr. Wendell L. Hughes, Hempstead, N. Y.: I did not have an opportunity to review these papers before this evening. Speaking about anesthesias, I think that before injections are made through the conjunctiva, a pledget of cotton soaked in the local anesthetic and held in contact with the area in which one wants to pick up the conjunctiva and make the injection is useful in preventing any pain when one picks up the conjunctiva, which is usually not completely anesthetized by the instillation.

I should like to mention the subfascial injection of procaine, rather than the classical retrobulbar injection. Retrobulbar injection is made far back into the orbit; the subfascial injection is made by anesthetizing with a pledget, as above, and lifting up the tendon of the superior rectus with a 5/8 in. No. 27 needle, injecting beneath the superior rectus muscle 1.5 cc. of procaine hydrochloride with epinephrine. This method has given very quick, thorough anesthesia without the addition of hyaluronidase (Hydase). The hyaluronidase, of course, makes the procaine effect wear off a little more quickly. This injection is useful because it avoids the possibility of a retrobulbar hemorrhage, there being no vessels in that area; and in the eight or nine years I have been using it I have not seen one retrobulbar hemorrhage. When that occurs, it is rather distressing both to the patient and to the surgeon. That complication, I think, can be avoided. The anesthesia is quicker and more certain with the subfascial injection. The anesthetic seeps around the posterior part of the eye and catches the nerves as they enter the sclera from behind. I think the anesthesia is much more certain and quicker and lasts well through the operation.

Dr. McLean dwelt on replacement of the iris, as did Dr. Guyton. A knuckle at the base of the iris is frequently present, held up in the wound by a little clot; and

if one lifts up the corneal wound and looks at it, one will be surprised how frequently there is a knuckle of iris at the base, which is against the scleral portion of the wound. This can easily be separated with a small iris spatula before the lens is removed. If it is not removed, it is likely to stay there after the operation is completed and lead to a drawn-up pupil.

As to the question of iris prolapse, I think when atropine is used postoperatively iris prolapses are much more frequent than when a miotic is used. Even in cases in which a complete iridectomy is done, a miotic helps to contract the remaining portions of the sphincter and pull the iris pillars down out of the wound, preventing a knuckle of iris from being incarcerated in the wound. A small knuckle at the base of the iris is just as important as a complete pillar being out in the wound.

Both Dr. McLean and Dr. Guyton are, from their remarks, adherents of the use of the capsule forceps. I like the suction erysiphake, for several reasons: If one slips off the capsule after grasping it with the capsule forceps, one usually takes a piece of capsule with it. If one slips with the erysiphake, one can go back almost any number of times, put it back on, and eventually get the lens out, if necessary, after five or six attempts. One can usually lift the lens from the anterior surface of the vitreous and pull it out, with very little danger of rupture of the capsule. Of course, some pressure from behind helps in extracting the cataract without too much tension on the lens.

There are a number of complications that neither Dr. McLean nor Dr. Guyton dwelt upon, the first being the complication incident to the removal of sutures. That can be dealt with briefly by saying that it can be eliminated by the use of chromicized absorbable surgical (gut) sutures, as the sutures come out spontaneously and there is no trauma or accident incident to their removal. There are complications incident to the putting in of the gut sutures because of their being not as flexible or as easily visible as silk sutures; but if one puts in gut sutures by a technique whereby the suture does not bridge directly across the wound, it can be done quite readily, without their slightly increased rigidity interfering with the extraction.

Dr. Dunnington has shown that when there is an epithelial margin of conjunctiva near the wound it is very prone to grow down into the wound. When that occurs, one is likely to get an iris cyst or an epithelial downgrowth on the posterior surface of the cornea. I feel that these complications are due to invasion of conjunctival epithelium into the wound. They can be avoided by not using direct corneoscleral suture or any suture in which the edge of the conjunctiva attached to the cornea is included in the suture. Dr. McLean's type of suture avoids this complication, and that feature is rather important. The knot of a gut suture should not be placed over the cornea. The McLean type of suture is not well suited to gut. Therefore other types of sutures can better be used when the knot is to be placed over the conjunctiva, and as the hard knot nestles into the conjunctiva, it will not traumatize the cornea.

Of the recently publicized features of cataract work, Dr. Guyton mentioned use of the Ridley implant; another is the injection of lens extract. The latter substance, fish lens extract, has been used since 1937, and human lens extract was used many years before that. The extraction of a cataract after the injection of lens extract—and I recently removed one that became thoroughly mature after six weeks of intensive treatment with fish lens extract—has been done without postoperative complications. In the case of which I speak the lens was very mature and rather swollen; so I elected to do an extracapsular type of extraction and wash out the soft lens material. It all washed out well, and the eye healed nicely. We have been warned about sensitivity to lens proteins in these cases. It may be that in a large series of cases sensitivity will occur, but I have not found it, and I have talked to others who have had the same experience.

Dr. Guyton showed vitreous loss in his picture. A type of suture that can be drawn up immediately behind the emerging lens, shaving off any vitreous that may be attached to the lens capsule by closing the wound directly, is to be recommended.

When postoperative glaucoma is to be feared, I have preferred to plan an intracapsular extraction with an anterior sclerectomy and iris inclusion at the same time. This procedure has been done 25 times, with almost universally good results when glaucoma has been present with the cataract.

I should like to ask of the essayists what they do when the epithelium becomes slightly edematous, with usually a rather prolonged reaction in the eye, with some clouding of the stroma and occasional wrinkles in the posterior surface of the cornea. Such reactions have been particularly bothersome to me, and I should like to know what procedures they have used in such cases.

A small knuckle of vitreous in the wound is just as important as a lot of vitreous, as it affects the healing of the wound, even though the hyaloid membrane is still intact. It is well to make a notation on the operative record that there is a knuckle of vitreous in the wound and a statement as to whether the hyaloid membrane is intact or not, in order that these eyes may be followed more accurately. The presence of vitreous in the wound, whether it is a great deal or a little, does delay the healing somewhat, but it is surprising how some eyes with extra loss of vitreous will heal well, with good vision.

Regarding the structure of the vitreous which Dr. Guyton described, in a recent article in the *British Journal of Ophthalmology* are reported the results of examination of the vitreous with the electron microscope with extremely high magnifications, showing definite fibrillar and nodular structure of the vitreous with variations in structure in different species. I should like to ask Dr. Guyton whether he feels that the contact of vitreous on the posterior corneal surface has anything to do with the production of epithelial edema and dystrophy.

In putting air in the anterior chamber, I think it important to have the head of the bed raised about 30 degrees, with the head of the patient on a pillow, so that the eye is in such a position that the air bubble tends to insert itself along the inner margin of the wound. If the air is put in and the patient kept flat, it is worse than putting in no air at all, because the air displaces the iris and other structures up toward the wound; but if the patient is on a pillow, with the head of the bed up and the eye in such a position that the air inserts itself behind the wound, the air tends to separate the structures of the interior of the eye from the wound itself.

I should like to ask Dr. Guyton what he does for hemorrhage into the vitreous, whether there is any treatment with which he has been able to clear the clot in the vitreous. It is interesting to conjecture about the use of hyaluronidase in these cases, which, if given subconjunctivally, will hasten the absorption of subconjunctival hemorrhage.

Have the essayists had any experience with therapeusis of hyaluronic acid or hyaluronidase in opacities of the vitreous and separation of the vitreous? Is there any similar effect on hemorrhage into the vitreous? Also, what does Dr. Guyton do for infection of the vitreous? We formerly had it, but in recent years it has been entirely avoided by the injection subconjunctivally of 50,000 units of penicillin in the region just back of the wound. Both von Sallmann and Sorsby have shown that penicillin given intramuscularly is not absorbed into the eye, but if given subconjunctivally, it will have a definite concentration in aqueous and vitreous and therefore help in preventing postoperative infection. There have been no infections in the past five years in any intraocular operation I have done, I believe solely because of the subconjunctival injection of penicillin.

Concerning Dr. Guyton's statement about the depth of focus in cases in which there is a small pupil, I am wondering whether that assertion is based on actual measurements or not. I do not know of any series of cases in which the depth of focus actually has been tested for comparative purposes.

Dr. Constantine remarked on the use of corneal lenses, and I am wondering whether she has measured any of the eyes for aniseikonia afterward. It would be interesting to know just how much aniseikonia is induced by the correction of aphakia in a previously emmetropic pair of eyes, and whether the difficulty in vision afterward might not be due to a rather high degree of aniseikonia. If the image of

the eye in which aphakia has been corrected with the corneal lens is greater in size than that of the emmetropic eye, it would be reasonable to assume that the difference could be corrected by a corneal lens, overcorrecting the hypermetropia by 1 or 2 D. with the addition of a —1 or 2 D. lens in front of that to minify the size of the image. That would have to be measured in specific cases to determine the exact manipulation of the "size" conditions necessary.

Was the epithelium examined in the case in which autopsy was done with the corneal lenses in position? I am interested to know whether the long wearing of the contact lenses would tend to wear down the corneal epithelium or whether it would produce a sort of keratinization or thickening of the epithelium as a defense reaction on the part of the cornea.

DR. JACK S. GUYTON, Baltimore: I question a few of the points Dr. McLean mentioned. First, in cases of long-standing diabetes, or others in which there is a particular tendency to bleed, and in cases of filtering operations extending a little way into the cornea, I am far from convinced that a shallow, purely corneal incision is the incision of choice. Healing is more rapid if the incision is placed so that the superficial fourth of it is in vascular tissue and the inner three-fourths in clear cornea; and if coaptation of the incision is perfect, with at least three corneoscleral sutures, I do not believe one will get appreciably more anterior chamber hemorrhages than with the incision placed within clear cornea.

Second, I question the advisability of radial iridotomy at the time of cataract extraction except in very rare cases. If the pupil does not dilate well, it easily can be stretched rather than necessitate radial iridotomies, and I do not see the point in having a large pupil. The smaller the pupil, within limits, the better will be the effective vision in that there will be a greater depth of focus.

Third, with regard to cataract extraction in eyes with chronic uveitis, I think that it is most unlikely to do any damage to such an eye, or to stir up an inactive uveitis, even if corticotropin or cortisone is not used (unless the uveitis is a sympathetic ophthalmia).

DR. JOHN M. MCLEAN: I am grateful to Dr. Hughes for having brought out a number of complications which we did not cover. It was not because we were unaware of them or because we have not had them. I think that, between the two of us, Dr. Guyton and I have had every complication in the book, and probably a few that are not in the book; but we did not have time to cover everything in the time allotted by the chairman. Dr. Hughes has covered a number of additional complications of cataract surgery very ably. All I can say is that essentially I endorse what he has said. Perhaps I can answer for Dr. Constantine, since the patient who wore the contact lenses for approximately 10 years before death was originally a patient of mine. We did not have sections of the eye at autopsy, and we cannot answer the question as to what the state of the corneal epithelium was histologically. On the biomicroscope it appeared essentially normal up to the last time she was examined.

Dr. Hughes mentioned the use of miotics at the end of extraction. That is something which should always be considered, particularly if the pupil is round; but also, when a combined extraction has been done, miotics are in order if the extraction has been intracapsular and the pupil is large at the end of the operation. Sometimes manipulation of the iris in itself causes sufficient tissue reaction to produce the normal tissue miotic (histamine), and a pharmacological supplement does not seem to be in order. It is probably of not great importance whether a miotic, mydriatic, or any agent at all is used at the end of extraction. In my average uncomplicated extractions I do not instill any drug at the end of the operation and the course seems to be satisfactory.

Dr. Hughes spoke of subfascial injection instead of retrobulbar injection. I have tried this but prefer the latter. Subfascial injection does give good anesthesia, as he says, but I do not believe it gives as good akinesia of the rectus and oblique muscles as a properly placed retrobulbar injection does; if I were to substitute something for retrobulbar injection, I should prefer curare, but in the ordinary case I use retrobulbar injection.

Dr. Guyton mentioned the question of purely corneal section in bleeders. I am well aware of the fact that capillaries tend to bridge the corneal wound and they may bleed; but I still prefer not to cut across any more blood vessels in patients who tend to bleed than is absolutely necessary, and I still think I get better results if I make purely corneal sections in those patients who tend to bleed, and if I close them tightly with corneal sutures.

Dr. Guyton raised a theoretical question about the size of the pupil and the depth of focus, which Dr. Hughes has already commented on; his third remark had to do with uveitis, lens extraction, and adrenocortical agents. When it is essential to remove a lens, and sometimes it is in an eye which is violently inflamed, I feel happier, and I think the eye does better, if the inflammatory reaction can be reduced before the operation is started; at the moment, I think the most efficient means of suppressing that inflammatory reaction is through the use of adrenocorticotropic agents.

Dr. Elizabeth F. Constantine: In reply to Dr. Hughes's question, we have not measured the aniseikonia in these patients; that is something we hope to do as we go on with this series.

Dr. Jack S. Guyton, Baltimore: Dr. Hughes, in his very worth-while and condensed summary of the many points we left out, brought up, among other things, the question of corneal edema: Is it due to vitreous against the back of the cornea or not? That, I think, is a very important point, and I can only give my personal beliefs. I believe that when vitreous presses against the posterior surface of the cornea, if only the normal fibrillar sheets of the vitreous are in contact (i. e., no "thickening" or fibrosis), there is unlikely to be any appreciable interference with the dehydrating mechanism of the cornea. However, if the incision is placed rather anteriorly and if, during the postoperative course, the anterior chamber should get a little shallow, with the pupil widely dilated and the vitreous bulging up through it, there is a pretty good chance that vitreous will stick to the incision. If it does stick in the incision, we know that fibroblasts are going to grow along the vitreous sheets and thereby result in "condensation" of the surface of the vitreous. In such a case, as well as with "condensation" of the anterior hyaloid membrane from other causes, there is considerable likelihood of corneal edema if the vitreous surface presses on corneal endothelium. Thus, if the iris sticks to the vitreous, with some growth of fibroblasts from the iris onto the surface of the vitreous—again, what we often call "condensation" on the surface of the vitreous, even when it has nothing to do with the site of operative incision—and if we get what Dr. Chandler emphasizes so commonly, a plug of vitreous bulging through the pupil, then even if glaucoma should not ensue, corneal edema probably would.

For the treatment of corneal edema when vitreous is in contact with cornea, I should first try the injection of air. If vitreous and cornea do not separate, as is probable, one should try to separate them with a cyclodialysis spatula. I am not sure how much good that does in restoring the barrier unless the condition is just on the borderline.

With the use of hyaluronidase to liquefy vitreous I have had no personal experience. Dr. Schimek, at Wilmer Institute, tried various concentrations in an attempt to cause liquefaction of the rabbit's vitreous, and he found he could get apparent liquefaction of the vitreous structure without histologic evidence of retinal damage, but the concentration he had to use was between one-fourth and one-half that which did give histologic retinal damage; the range seems too narrow for practical use. For absorption of blood in the vitreous, hyaluronidase in a much lower concentration might theoretically be of some benefit.

In regard to the depth of focus with the small pupil and the large pupil, I have noticed time after time in refracting aphakic eyes that one with a very small pupil can attain 20/15 or 20/20 vision over a considerable range of spherical correction, whereas for an eye with a large pupil there is a rather critical correction. Simple optics will show why this is true.

A final point regarding prevention of loss of vitreous: Pressure should be applied over the closed lids for five minutes immediately after the retrobulbar injection.

News and Comment

GENERAL NEWS

Certification as Honorary Master in Ophthalmic Optics.—The American Board of Opticianry is continuing certification as Honorary Master in Ophthalmic Optics during the year 1954. In 1947 the Board established honorary certification on the basis of the records and years of experience of the applicant, and earned certification through the examination of graduates of schools maintaining an acceptable two-year collegiate curriculum. A prerequisite for honorary certification is that the candidate must have been actively engaged for 15 or more years in manufacturing, prescription, laboratory work, or optical dispensing. At the present time 1,607 persons have been certificated as Honorary Masters in Ophthalmic Optics. Sixty-eight (68) graduates of the schools, the first class being graduated in 1948, have been certified as Associates in Ophthalmic Optics and have received the degree of Associate in Arts at the Los Angeles City College; the Institute of Applied Arts and Sciences, Buffalo, N. Y., or Rochester Junior College, Rochester, Minn.

Communication Aids for the Blind.—A session on Communication Aids for the Blind will be held at the 120th meeting of the American Association for the Advancement of Science on Dec. 28, 1953, in Boston.

Better ways for the blind to read, walk, and communicate will be discussed by research authorities in the field at a symposium on Communication Aids for the Blind to be held in conjunction with the meeting of the Association.

Wayne University Postgraduate Course.—The Department of Ophthalmology of Wayne University College of Medicine will give nine months' postgraduate training in basic ophthalmology, beginning Sept. 13, 1954.

Six mornings a week are spent in lectures and laboratories. Each afternoon students are assigned to eye clinics of affiliated hospitals. A fundus clinic is held weekly; also, one on muscles.

Students are given 120 hours of training in physiological optics, 122 hours in histology and pathology, 60 hours in biochemistry, 40 hours in neuroanatomy, etc.

Tuition is $900. Brochures on the basic science course will be mailed upon request. For further information, write Dr. A. D. Ruedemann, chairman, Department of Ophthalmology, 690 Mullett St., Detroit 26.

PERSONAL NEWS

Beverly Myers Nelson Achievement Awards.—At the meetings of the Education Foundation in Ophthalmic Optics of the American Board of Opticianry, Beverly Myers Nelson Achievement Awards, created by Leslie W. Myers of Minneapolis as a part of the Foundation, were presented to William L. Benedict, M.D., of Rochester, Minn. and to Jack C. Copeland, O.D., of Chicago. Mr. Copeland has been for many years technical consultant in ophthalmic optics to the Bausch & Lomb Optical Company and is known for several contributions of methods and apparatus in the field of refraction. Dr. Benedict, secretary of the American Academy of Ophthalmology and Otolaryngology, was honored because of his broad interest in and support of education and research in all phases of vision and ophthalmic optics.

Books

Ophthalmologic Diagnosis. By F. Herbert Haessler, M.D., Professor and Director of the Division of Ophthalmology, Marquette University School of Medicine. Price, $8. Pp. 387, with 151 figures. The Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1953.

This new book is divided into two distinct and overlapping parts. As the author states in his preface, "The first five chapters are offered as an example of diagnostic procedure that is based on an abbreviated list of manifestations. The remainder of the book is a series of essays in which further comment is elaborated to elucidate the situations in which it seemed to me that help would be welcome." The titles of the respective chapters are as follows: The General Survey of the Patient, The Disturbances of Functions, Manifestations of Abnormalities in the Adnexa, The Anterior Segment, Media and Fundus, and, in the more detailed chapters, Functions, Adnexa, Anterior Segment, and Posterior Segment. The term "essays" most aptly describes the general tone of the book. At times the author assumes the attitude of a teacher talking to a beginner student, and at times a philosophical overtone which might properly be directed to a lay audience. The author approaches the problems in diagnosis as representing abnormalities in various functions of the eye. There are many practical hints in the observation and interpretation of clinical findings which have stood the test of time and have proved to be of value in the author's experience in ophthalmology.

The author's greatest interest is apparently in the morphology of cataracts, to which he devotes 31 pages; a discussion of changes in the disk and visual fields, and their correlation with lesions in the optic pathways, and a discussion of intraocular tumors. However, it is somewhat disappointing to find so little attention devoted to several important current problems—for example, only two pages on glaucoma and eight pages on uveitis. To the reviewer, some of the points in clinical differentiation of various forms of uveitis appear dubious, and the author does not discuss more recently described causes of uveitis, such as Toxoplasma, fungi, and viruses. Laboratory studies usually employed in the diagnosis of cases of uveitis, skin tests, and their interpretation are not mentioned. The discussions of muscle paralysis and head tilting are confusing. No explanation is given of the after-image test, and details which might be of value in the diagnosis of fusion anomalies are omitted. The concepts and differentiating points in the diagnosis of retinal vascular pathology do not bring in much of the more recent discussion of these conditions. As he states in the preface, none of the illustrations might be mistaken for the portrait of a sick eye. Ultrasimplified line drawings have been utilized, many of which are not sufficiently labeled, and the legends are at times lacking or inadequate. There are only a few references to the literature, most of which have been selected on a historical rather than a current basis.

Although many parts of the book are interesting because of the personal comments, historical allusions, and personal philosophy of the author, it is difficult to determine to what reading audience this book is directed. Its somewhat rambling discussion, emphasis on morphologic aspects of purely ophthalmological interest, and lack of recent advances in medical ophthalmology do not make it especially suitable for medical students. For the practicing ophthalmologist, there seems to be insufficient detail in the techniques and interpretations of modern diagnostic methods, including laboratory studies. It probably should be read by the mature ophthalmologist much as a novel is read, to attain a perspective into the philosophy and certain specific techniques of the author.

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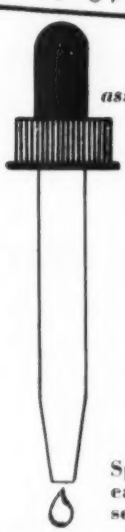

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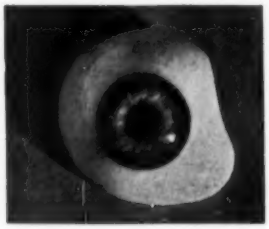

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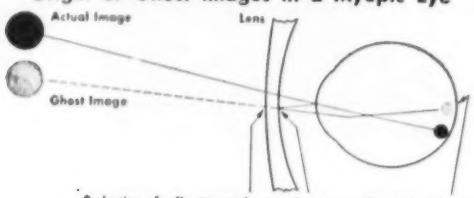

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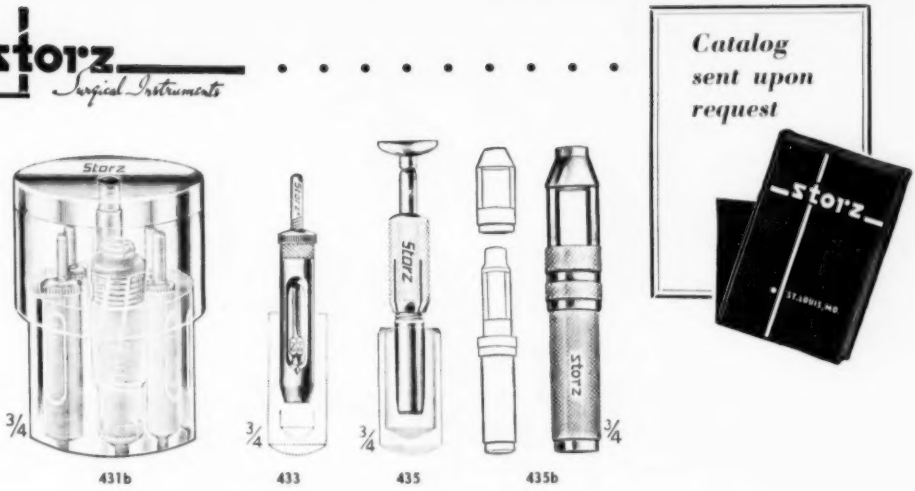

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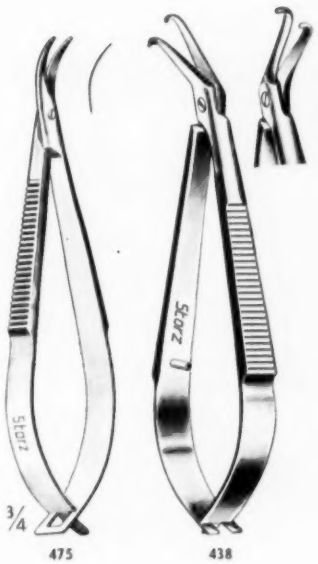

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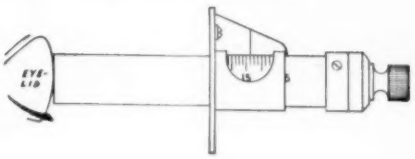

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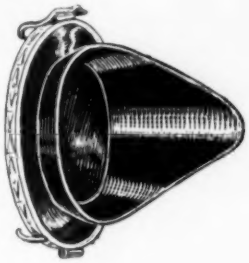

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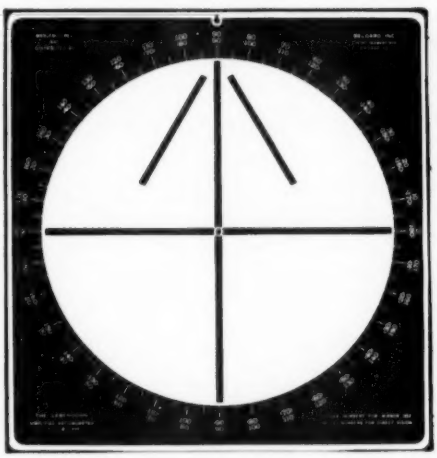

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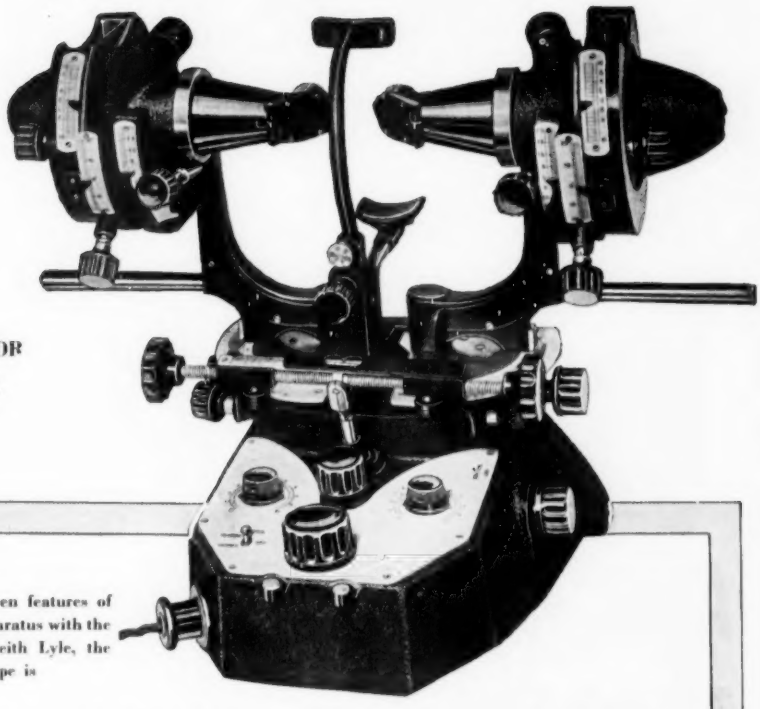

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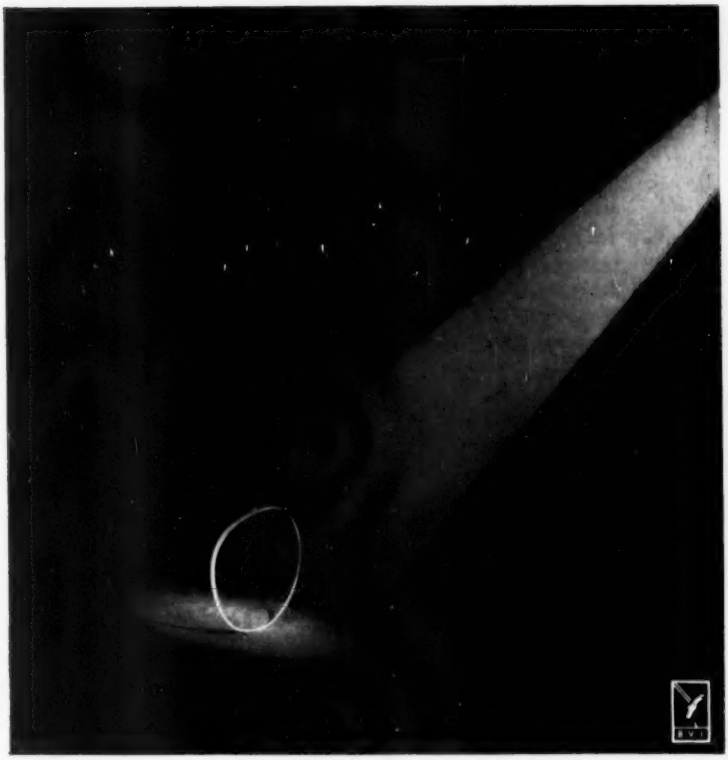

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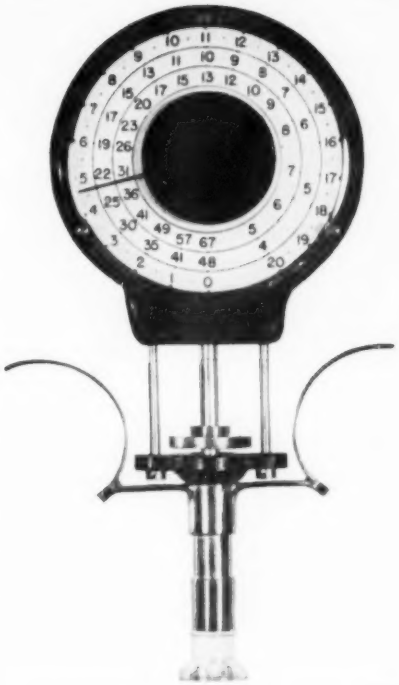

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